

PSYCHOPHYSIOLOGICAL STUDIES OF SLEEP AND DREAMS

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SUMMARY

Preface. The thesis comprises two separate studies, which differ not only in subject matter but also in methodology. The inclusion of two distinct studies is regarded as appropriate in a thesis representing the results of a research training. The first study sought to clarify a factual issue: could habituation, an elementary learning process, occur in human sleep? The second study is an instance of hypothesis testing: do certain drugs affect dream content in the manner predicted?

Chapter 1. This gives an introduction to sleep. It describes briefly the characteristics of REM (rapid eye movement) and NREM (non-REM) sleep. The different behaviour of the autonomic systems in these two kinds of sleep is described. A brief historical review of the relationship between REM sleep and dreaming is included.

Chapter 2, comprises a review of literature on discrimination, conditioning and learning during sleep.

Chapter 3, describes orienting, defensive and adaptation reactions in waking. It also includes a discussion of the small amount of theory surrounding the habituation of orienting reactions. The second part of this chapter discusses responses to novel stimuli presented during sleep. The third section describes problems and possible artifacts in the measurement of electrodermal and cardiac responses to stimuli.

Chapter 4, is a discussion of previous work on habituation in human sleep. Early studies, largely qualitative, mostly concluded that habituation was possible in sleep. Only two studies existed which examined each sleep stage separately. One of these concluded that habituation was not possible in sleep. This study, however, suffers serious methodological flaws which have not been recognised. The/

/The purpose of the present experiments was thus to remedy these flaws, and establish whether habituation of autonomic and electroencephalographic (EEG) responses was possible in human sleep.

Chapter 5, describes the design. Two autonomic responses - skin potential and heart rate responses - were monitored in each stage of sleep. EEG responses were also studied in REM sleep and in stage 2 NREM sleep. Possible habituation of these responses to repetitive tone stimuli was tested, as a function of the interval between stimuli, in sleep stage REM, in NREM stages 2 and 4, and in daytime controls.

Chapter 6, is a discussion of methodological problems, including subject selection and data analysis.

Chapter 7, details the actual procedures used.

Chapter 8, describes the results. Habituation of the skin potential response occurred under all conditions. Heart rate responses habituated in NREM sleep, but did not habituate either in the daytime controls, when subjects were drowsy, or in REM sleep. EEG responses in both REM and NREM sleep demonstrated habituation, but for these responses habituation was markedly affected by interstimulus interval.

Chapter 9, discusses the lack of heart rate response habituation found in drowsiness and REM sleep. It is considered possible that a lack of response habituation in some meditational states may be related to the lack of habituation found here in a state of drowsiness.

The different behaviours of the cardiac, electrodermal and EEG response systems are regarded as important. It is concluded in/

/in this chapter and in chapter 10 that differences between the behaviour of the various responses comprising that "orienting reaction" have been overlooked too long. The whole concept of "an orienting reaction" is questioned.

Part B. The Effect of drugs on dreams.

Chapter 11, describes the effects of various psychoactive drugs on REM sleep. Most drugs which do affect REM sleep reduce or suppress it, whether they are stimulants or depressants. Barbiturate and non-barbiturate hypnotics which suppress REM sleep also reduce the profusion of eye movements (EMs) in REM sleep.

Chapter 12, reviews the literature relating the physiology of the REM period to the content of dreams derived from REM periods. It is concluded that the evidence is insufficient to support the idea that EMs in REM sleep are related to the dreamer's shifts of gaze. There is, however, evidence for some relation between the 'intensity' of a dream and the 'intensity' of the REM period. There is also evidence that "phasic events" during REM sleep are associated with the appearance of bizarre elements in dream reports. Differences between thought-processes and dream-processes in REM and NREM sleep are discussed. The chapter also includes a discussion of the relation between REM sleep and nightmares.

Chapter 13, reviews previous studies of the effects of drugs on dream content.

Chapter 14, describes the purpose of the present study. This was to test the hypothesis that since barbiturate and non-barbiturate hypnotics can be said to make REM periods less 'intense', they should/

/should also make dreams less 'intense', given some relation between the physiology of the REM and the dream content.

Chapter 15, describes the various approaches possible to the quantification of dream content.

Chapter 16, details the method. This involved subjects on either placebo, amylobarbitone or nitrazepam for periods of baseline, 'drug', and withdrawal.

Chapter 17, gives the results. The hypothesis was not supported. Effects of the drugs on the dreams were scarce, while REM sleep was drastically affected. However, two nightmares occurred in withdrawal.

Chapter 18. The discussion of the results stresses the variability in individuals' subjective responses. It is suggested that personality may interact with the drug, and that this effect is a more important influence on dream content than the physiology of the REM period.

It is concluded in chapter 19 that dreaming in REM sleep can be regarded as part of a continual, 24 hour, mental activity. This is governed by the level of cortical arousal. In particular, mental activity during REM sleep may be interrupted by the "phasic events" which are also associated with bursts of EMs. Relationships between EMs and dream content are therefore only peripheral.

and

PREFACE

This thesis is divided into two parts, and in this respect it is somewhat unusual. Many theses consist of a series of separate studies which the author relates in some way. However, I have not attempted to tie together the two studies in this thesis. Research for a higher degree is often not treated as a training but as one's first venture into research. I believe it should remain primarily (though not solely) a training in research. In this frame of reference I trust that the two parts of this thesis will not be too great a surprise for the reader. Part A (Habituation in sleep) was essentially an exercise in establishing a point of fact - whether an elementary learning process could occur in sleep. Part B (The effect of drugs on dreams) was a study conceived quite differently: it was done to test a hypothesis which had been only briefly and insufficiently examined in an earlier study. Thus in certain ways the two studies are complementary. The first had clearly defined objectives and hence only a few dependent variables. The second involved a search for effects, and hence a more extensive examination of a variety of dependent variables. In this light the reader may be able to understand why I have chosen to present two essentially separate studies under one cover.

Some of the material in Part A is being published (Firth, 1973a). The results of the first experiment in Part B are also published (Firth, 1973b); the final results, however, have yet to be published.

ACKNOWLEDGEMENTS

Many people have helped me to clarify what I have set forth in the following pages. Help in sorting out one's ideas comes from many conversations with a variety of people, and most of these contacts are very informal. I especially wish to thank Dr. Ian Oswald (my supervisor), who has always been especially vigilant when I have shown evidence of thinking loosely, ambiguously, or quite simply inaccurately. He has also put some of my ideas into very proper perspective. For my part I have tried to disabuse him of some fondly held ideas about the effects of drugs on dreams. If I have made him sceptical of one notion he used to hold, he has forced me to be sceptical of many things I might have believed, which is right and proper, if a little uncomfortable at times for me. In particular I should like to thank him for his careful reading of, and his comments on, this thesis.

Stuart Lewis gave me a lot of help at the time I most needed it, when I had only recently arrived. He and the other members of the laboratory have all listened to me at times, for which I appreciate them - Des Dunleavy, Susan Allen, Joanna Tagney, Vlasta Brezinova, Alistair MacLean, Marion Briggs, and Chris Smith have all responded to my ideas in one way or another, however informally.

Special help in analysing my data, for which I am very grateful, came from two people. David Williams of the Department of Statistics helped me with computing in the first of these studies. Alistair MacLean, among other things, expended time and energy helping me design and analyse the second of the two studies.

I am very much indebted to Dr. Aitken for the loan of equipment for the continuous recording of heart rate, for the experiments in the first part of the thesis.

Several people have performed a tangible service by scoring data. Dr. Oswald scored many EEG responses for the first of these studies, and he read every dream I collected in the second of the studies. Fruitlessly, he tried to guess on which night each dream was collected. I trust he does not regret the time he spent doing so! John Trinder of the VA Hospital, Cincinnati also gave a lot of time to scoring all the dreams collected from a dozen subjects. It is their results which enable me to say with confidence that my conclusions are not a result of my own preconceptions.

I also wish to thank our librarian, Margot Bonar, for dealing with my voluminous library requests.

My subjects naturally deserve thanks for putting up with the indignities involved in being a guinea-pig for sleep research. My compliments especially to those who unprotestingly told their dreams on being so rudely awakened in the midst of the night.

My vigilant supervisor has also helped to make this thesis more readable, by correcting some of my worse grammatical errors, and spelling mistakes. Barbara Bierer rashly offered to read one or two chapters and was then cruelly tricked by me into proof-reading the rest. She too has tried to repair my failing grammar, to the point where I am almost scared to write another sentence. I am grateful to her despite this.

- iiia -

Christina Robb has very kindly typed this for me. I hope she has not become as involved in it as I have at times. My friends - especially my flat-mates - have had to put up with a lot from me at times, not least at breakfast, hearing my recurrent dreams about my thesis. At times they have actually been quite understanding.

Without whom all the poeple I have talked to at conferences ... and at parties too.



Eden Grove

Bond

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GLOSSARY

Explanation of Abbreviations used

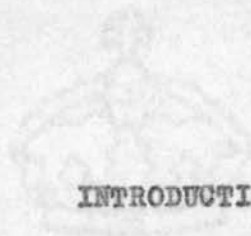
REM sleep	- rapid eye movement sleep
REMP (s)	- rapid eye movement sleep period (s)
EM (s)	- eye movement (s)
NREM sleep	- sleep other than rapid eye movement sleep
SWS	- slow wave sleep (stages 3 and 4)
EEG	- electroencephalographic, electroencephalograph
EMG	- electromyogram
EOG	- electro-oculogram
E	- experimenter
Hz	- cycles per sec
OR (s)	- orienting reaction, response, reflex.
DR (s)	- defensive reaction, response, reflex.
HRR (s)	- heart rate response
SPR (s)	- skin potential response
GSR (s)	- galvanic skin response (skin resistance response)
EDR (s)	- electrodermal response (either SPR or GSR)
FPR (s)	- finger plethysmographic response
ISI (s)	- interstimulus interval
ANOVAR (s)	- analysis of variance
SS	- sum of squares
MS	- mean square
DF	- number of degrees of freedom
p	- probability of obtaining results by chance
PGO	- pontogeniculo-occipital
PIP (s)	- phasic integrated periorbital activity
SCE	- secondary cognitive elaboration
PVE	- primary (primarily) visual experience

Classification of sleep stages:

Loomis et al (1937)		Rechtschaffen and Kales (1968)	
A		W, wakefulness	
B)		REM sleep	
	OR	NREM sleep stage 1, drowsiness	
C		NREM sleep stage 2	
D		NREM sleep stage 3)	SWS
E		NREM sleep stage 4)	

"One would hardly have thought from the psychological journals of 1930 to 1960 that a dominant feature of human beings was their tendency to lie down every 24 hours and become completely unresponsive (sic!) for seven or eight hours".

(Broadbent, 1971, p28)



INTRODUCTION

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CHAPTER ONE

SOME PRELIMINARY INFORMATION ABOUT SLEEP

1. REM and NREM Sleep

Aserinsky and Kleitman's observations (1953) that there are times during both babies' and adults' sleep when their eyeballs move about beneath the closed eyelids, and if woken then the adults will often tell a dream, has resulted in an explosive growth in research into sleep in the past twenty years.

Research following Aserinsky and Kleitman's (1953, 1955) emphasised two kinds of sleep - sleep without eye movements (EMs), and sleep with eye movements¹: rapid eye movement, paradoxical or simply REM sleep, which was seen as being uniquely associated with dreaming. Prior to Aserinsky's observations, sleep had been classified according to the system of Loomis et al (1937). Loomis et al divided sleep into stages A to E on the basis of the electroencephalograph (EEG). Stage A (drowsiness) contains some alpha activity (8-12 Hz). Stage B (drowsiness or light sleep) contains no alpha; the EEG is of low voltage mixed frequency with some 3-7 Hz activity often prominent. Stage C contains spindles (12-14 Hz), and K complexes - arousal reactions consisting of one or more high voltage slow waves with 12-15 Hz activity superimposed. Stages D and E contain increasing amounts of high voltage slow waves (1-2 Hz). Dement and Kleitman (1957b) preferred to assign numbers instead of letters to these sleep stages. Loomis' stages A and B were combined into/

¹The eye movements referred to here are rapid, conjugate movements, not the slow 'rolling' eye movements often observed during drowsiness at the onset of sleep.

/into stage 1, and stages C, D and E were denoted stages 2, 3 and 4, Aserinsky and Kleitman had noted that the rapid eye movements occurred in conjunction with a low voltage EEG. Dement and Kleitman hence dubbed the sleep with rapid eye movements 'emergent stage 1' - distinguishing it from 'descending stage 1' at the onset of sleep.

Sleep was thus seen as comprising 5 stages: stages 1 to 4, and stage 1 emergent, 1-REM, or just REM (see Fig. 1). However, it came to be seen that there were profound physiological differences between REM and non-REM (NREM, or orthodox) sleep, and the view has grown that there are "two kinds of sleep" or two "states" (Oswald, 1962b; Snyder, 1963). They have even been called the S- and D- states: Sleep and Dream states, with synchronised and desynchronised EEG, i.e. NREM and REM sleep respectively, (Hartmann, 1967b). Reviews of the physiology of the two kinds of sleep have been provided by Berger, 1969, Jouvet, 1967, Hartmann, 1967, Oswald, 1969, Shapiro, 1967 and Snyder, 1969. Briefly, REM sleep is associated with profound loss of hyoid muscle tone (Berger, 1961), brain temperatures and blood flow which are as high or higher than in waking (Kawamura and Sawyer, 1965; Baust, 1967), variable and high heart rate and respiration rates relative to those in NREM sleep (Snyder et al 1964).

Penile erections are also noted in REM sleep, but are rare in NREM sleep. Patterns of discharge of single neurons are different in REM and NREM sleep (Huttenlocher, 1961; Evarts, 1969).

Although we are far from understanding the mechanisms of REM/

/REM and NREM sleep with any certainty, they appear to be controlled by two distinct systems neurophysiologically. Thus, a cortex is necessary for the appearance of EEG slow wave activity (though behavioural sleep can occur in the decorticate cat). If a lesion is made in the dorsolateral pons, REM sleep does not occur, but NREM sleep is unimpaired (Jouvet, 1967, 1969). There is disagreement about the biochemical control of sleep, but it does seem that biochemically speaking as well as neurophysiologically speaking sleep is of two kinds, REM and NREM, and not merely a continuum of depth.

Recently certain hormonal changes have been linked to the 'two kinds' of sleep. Human growth hormone (HGH) is released largely in the early hours of sleep, and its release appears to be related to the occurrence of slow wave sleep (Parker et al 1969). By contrast, testosterone release is apparently related to REM sleep periods (Evans et al 1971a, Evans et al 1971b).

2. Autonomic aspects of REM and NREM sleep

Reviews of particularly autonomic functioning in the "two kinds" of sleep have been presented by Snyder (1967, 1971). The occurrence of penile erections is probably the autonomic event which most clearly discriminates REM from NREM sleep. This association was first noticed by Oswald (1962, p142), and subsequently confirmed by Fisher et al (1965) and Karacan et al (1966). At least 30% of REM periods (REMPs), are apparently accompanied by erections, and although erections do occasionally occur in NREM sleep (other than immediately before or after REMPs), this happens only rarely unless REM deprivation is/

/is achieved by drugs or by waking, when erections may break through into NREM sleep (Fisher, 1966, 1967). Detumescence during REMPs appears to be related to anxiety in the dream content associated with the REMP (Karacan, 1966).

Aserinsky and Kleitman (1955) originally reported that both heart rate and respiration rates were higher in REMPs than in NREM sleep either preceding or following the REMPs, and this has been confirmed repeatedly since (e.g. Shapiro, et al 1964), along with the finding that independent of sleep stage both heart rate and respiration rate decrease from the start to the end of the night (Snyder et al 1964).

More striking than the slight rise in mean rates during REMPs is the increase in the variability of respiration and heart-rate in REM sleep, first noted by Jouvet et al (1960). Aserinsky (1965) noted a consistent relationship between bursts of eye movements in REMPs and variations in respiration rate, and Hobson et al (1965) also found that variations in respiratory rate were often associated with bursts of eye movements, although they did not report such a consistent relationship as did Aserinsky.

As has been mentioned, mean respiration and heart rates decline slowly through the night. REMPs alternate with NREM sleep roughly every $1\frac{1}{2}$ hours in man, NREM sleep consisting chiefly of stages 3 and 4 early in the night, with more stage 2 late in the night. It used to be a common assumption that stage 4 was the 'deepest' stage of sleep - such an idea readily stems from the fact that stage 4 sleep contains the greatest amount of slow wave activity of any sleep stage; furthermore, arousal thresholds are high in stage 4 (Rechtschaffen et al 1966). Heart rate, skin conductance and/

/and other autonomic measures have often been used as indicators of 'arousal' in waking subjects. If, however, heart rate and respiration rate decline during the night as the EEG progressively (if irregularly) lightens, it becomes clear that concepts of sleep 'depth' cease to be of much use. REM sleep is sometimes referred to as 'paradoxical' sleep because the EEG more nearly resembles that of an awake, alert organism which is behaviourally the reverse of alert. If we adopt the "obvious" concept of sleep depth - the layman's idea (how difficult is the sleeper to waken?) - then we see that REM sleep cannot be placed anywhere on a dimension of sleep depth. For to arouse the sleeper with a 'neutral' sound, REM sleep is as 'deep' as stage 4; but using a reinforced stimulus reveals that REM sleep can be a light stage of sleep (Williams et al. 1966). The sleeper will respond to a meaningful stimulus, but not to one that has no significance for him.

Skin resistance rises rapidly at the onset of sleep, and falls abruptly on waking. However, it is by no means clear that basal skin resistance shows any consistent changes within sleep in relation to sleep stage changes. While Hawkins et al (1962) reported that skin resistance rose during REMPs, this has not been confirmed - Tart (1967), for instance, failed to find any changes with REMPs; indeed, although he described a slow fall in resistance after the first hour of sleep (stages 3 and 4), he noted subjects who showed consistent rises all night.

There is widespread agreement on the other hand about the occurrence of spontaneous fluctuations in skin resistance and skin potential. These occur extensively in stage 3 and 4 sleep (slow-wave sleep or SWS), often in "storms" lasting continually for several minutes at a time (Oswald, Taylor and Triesman, 1959 ; Lester et al 1967), whereas such spontaneous fluctuations are very rare in REM sleep.

Johnson (1966) and Snyder (1967) have both reported that finger plethysmographic fluctuations are most frequent in REM sleep and least frequent in stage 4 sleep. Thus, cardiovascular variability is greatest in REM, and electrodermal variability greatest in NREM sleep.

3. REM sleep and dreaming

Following the early experiments of Aserinsky and Kleitman (1953), Dement and Kleitman (1957b), Dement and Wolpert (1958) and Wolpert and Trosman (1958), one of the important distinctions between REM and NREM sleep was felt to be that dreaming occurred in REM sleep but not in NREM sleep. This was seen as a fundamental distinction between the two sorts of sleep. Thus when in the early studies "dreams" were recalled after more than 80% of wakings from REMPs, and after only about 10% of wakings from NREM sleep, it was assumed that the dreams recalled from NREM sleep were fragments of dreams which had taken place during the previous REMP. Failures to recall dreams from REMPs were ascribed to interference by the waking process (or repression) rather than to a lack of actual dreaming. Dement and Wolpert (1958) remarked that recent studies appeared "to have conclusively established rapid eye movements as an objective criterion of dream activity".

This optimism was hardly surprising only a few years after Aserinsky's discoveries. The extensive explorations of those like Maury nearly a century before (1865) into the dreams that occur at the onset of sleep were regarded as something different. They were "hypnagogic hallucinations" (Maury's term); they were seen as something totally distinct from (REM) dreaming.

While further work after 1960 suggested more and more that REM and NREM were two distinct "states" physiologically, this assumption has been steadily weakened as regards the psychological differentiation between REM and NREM. Foulkes in 1962 published the results of a Ph.D. investigating dream reports from different stages of sleep. He found that some mental content ("mentation") was recalled from three quarters of all NREM awakenings, and that "dreams" were recalled on slightly more than half of wakings even from stage 3 and 4 sleep. Whether dreaming actually occurs during all stages of sleep is still at issue - sufficiently so for Foulkes to feel the need to defend himself against sceptics (Foulkes, 1967).

But early ideas about the significance of REM sleep as dream sleep have changed. In 1960 Dement reported the results of an experiment in which subjects were deprived of REM sleep by waking them from it. They became irritable, anxious, and one subject "quit the study in apparent panic". Since REM sleep significantly exceeded normal limits (17 to 25% of total sleep) on the recovery nights, and since control subjects woken equally often from NREM sleep showed no effects, Dement interpreted his results in terms of a "need to dream". However, subsequent ~~works~~ by Snyder/

/Snyder (1963), Kales et al (1964), Sampson (1966) and Agnew et al (1967) - reviewed by Webb (1969) + has minimised the psychological changes following REM deprivation.

The occurrence of a recovery process from REM deprivation by drugs which more than compensates for the lost REM sleep (Oswald, 1969c) argues that there is more to REM sleep than merely the "need to dream". The large amounts of REM sleep taken by kittens whose eyes are not even open in their first week of life (Valaix et al 1964) is another fact which looms large in the minds of those who are now dubious of the clear relationship which seemed to exist around 1960 between REM sleep and dreaming. The status of this relationship will be discussed more fully in Chapter 12. In particular, the way in which phasic events have come to be seen as the important constituents of REM sleep (and also NREM sleep) has considerably altered the tenor of ideas about the relation of dreaming to different aspects of sleep.

4. Patterns of sleep and measures of sleep

REM and NREM sleep alternate during the night. Except in some narcoleptics, who may pass directly into REM sleep at the start of the night (Rechtschaffen et al 1963b; Oswald, 1969b), sleep always starts with a period of NREM sleep. From wakefulness the subject passes through stages 1, 2 and 3 to stage 4; a period of SWS (stages 3 and 4) lasts typically about 50 minutes before there is a return to stage 2, usually with a brief period of REM sleep. The cycle is then repeated, with another period of SWS, and after about 90 minutes, another REM. As the night wears on, there is less/

/less SWS in the NREM sleep periods, and the REMPs become longer. At the start of the night, the first REMP lasts about 5 minutes or less, the second 10 or 15, the third about 20, and the fourth and subsequent REMPs may last around half an hour. The actual sleep pattern, however, tends to be somewhat more broken than this ideal picture would suggest. If each 20 second 'epoch' is scored as being in one and only one stage, then anything between 70 and 150 stage changes will occur in a 'typical' night, though this figure is subject to large individual differences.

The first measure that may be made of a night's sleep is the delay to sleep. This can be defined in various ways, but is conveniently taken to be the time between 'lights out' and the first stage 2 sleep (as defined by the internationally standard manual of Rechtschaffen and Kales, 1968). Some workers choose instead to use the time to the first stage 1 of the night. The choice is essentially arbitrary, but in subjective terms stage 1 is not perceived as 'sleep'. Thus Lewis (1969b) found that relative to EEG estimates of sleep onset as occurring at the first stage 2 of the night, subjects judged themselves to have taken even longer to get to sleep. Drowsiness at the start of the night was regarded by subjects as part of the "awake" period rather than the "asleep" period. This appears to be in contrast to subjects' perception of wakefulness during the night, for brief arousals. Thus Lewis's subjects who had not had medication, when asked to estimate how many times they woke during the night, gave a figure which agreed fairly well with the number of arousals to wakefulness/

/wakefulness lasting one minute or more.

Another convenient measure derivable from the EEG sleep record is the delay to REM sleep, from sleep onset. This measure ("delay to REM" or "REM latency") has a normal lower limit of about 45 minutes. Oswald and Thacore (1963) estimated that this value would be exceeded in all but one or two in every hundred records; Lewis (1969 p.14) confirmed this estimate. The delay to REM is however on occasion much longer than the typical 60 minutes. Sometimes the first REM is "missed", or to be more accurate, REM sleep itself does not occur at the end of the first cycle of SWS. A very brief period of stage 2, perhaps with arousals to stage 1, is followed by a return to SWS for another hour. In these cases the delay to REM sleep is not uncommonly as long as 150 minutes. This is especially liable to occur when drugs suppress REM sleep.

A variety of other measures of sleep can be extracted from a night of EEG record which has been scored into sleep stages epoch by epoch. The total sleep time (TST), total time in each of the stages, and percent (of total sleep time) in each of the stages are commonly used parameters of sleep. They have not been used in these studies, however, since with sleep interrupted or disturbed (subjects being woken up, or stimuli presented during the night), comparisons with normative data would have little meaning. / Other measures of sleep which can be derived from an epoch by epoch analysis involve stage changes. For instance, the number of changes to stage 1 or wakefulness can be used to assess how 'restless' sleep is.

Other parameters of sleep require a more detailed analysis of the night's records. For instance, small body movements may be counted from the record of muscle activity (EMG) - this provides another index of 'restless' sleep, which has been used in a study of the effect of a beverage on sleep (Březinová and Oswald, 1972).

One of the concepts which has gained importance in recent years is the distinction between phasic and tonic aspects of sleep, especially in REM sleep. Moruzzi (1963) first discussed the idea that as important a distinction as that between REM and NREM sleep was that between 'phasic' and 'tonic' aspects of REM sleep. The predominantly low voltage mixed frequency EEG is the most obvious tonic - i.e. persisting - aspect of REM sleep. The rapid eye movements themselves are the most obvious phasic - i.e. transitory - aspects. Bursts of eye movements are correlated with other 'phasic' events such as changes in respiration rate, plethysmographic responses, electrodermal responses, and periorbital phasic integrated potentials (PIPs) (Spreng et al 1968; Aserinsky, 1965, 1967a; Broughton et al 1965; Rechtschaffen, et al 1970). It would appear that PIPs indicate the occurrence of ponto-geniculate-occipital (PGO) spike activity in humans. PGO activity is apparently important in its own right, and it has been postulated that REM sleep in fact merely serves to allow the release of PGO activity (Dement, 1969). Whatever the merits of such a hypothesis, the experiments by Dement on the deprivation of PGO activity in the cat, which results in a much enhanced REM 'rebound' or compensation on recovery, do indicate that PGO activity is undoubtedly not a marginal aspect of REM/

/REM sleep (Dement, 1969).

Since PGO spikes are highly correlated with EM bursts in the cat (Bizzi and Brooks, 1963 ; Michel et al 1964 ; Jeannerod et al 1965 and Jouvet, 1969), and since also in man EM bursts are highly correlated with the nearest measurable variable - PIPs - it would seem that EMs are a good index of functionally important phasic activity in the human brain. The recording of PIPs in the human necessitates fairly sophisticated amplification procedures, but the recording of EMs does not.

EMs are normally recorded (as two channels of electro-oculogram) along with the EEG of a night's sleep recording. Counting the number of EMs occurring in a given REM is one way by which the "quantification" of REM sleep can be achieved (Lewis, 1968b). However, this procedure is extremely tedious, and so other schemes have been devised. The fact that eye movements tend to occur in bursts, instead of being uniformly distributed throughout REMs (Aserinsky, 1971) enables an estimate of EM "profusion" to be made by counting the number of short epochs which actually contain eye movements. Each epoch within the period scored as REM sleep is scored as either containing, or as not containing eye movement.

Such scores are to be distinguished carefully from the "density" of EMs: such a term refers to the number of EMs per unit time, i.e. it necessitates the counting of the actual number of eye movements as in the study of Lewis, 1968b. If the length of the epoch used for scoring is very small, and is comparable with the minimum interval between actual eye movements (less than .1 sec), then the profusion/

/profusion score approximates to the true density for that particular duration of REM sleep.

As the length of the chosen epoch lengthens, a profusion score ceases to be an indication of the number of eye movements, and becomes instead an indication of the number of EM bursts. Aserinsky (1971) has presented an excellent description of the distribution of EMs within normal REM sleep. The modal interval between successive EMs was around .5 sec. The distribution of EM intervals appeared to contain another peak just beyond 16 sec, which was interpreted as reflecting the mode for intervals between bursts. Calling EMs less than 8 sec apart part of a burst, the median burst length (i.e. duration) was $2\frac{1}{2}$ sec.

The use of epochs for scoring the presence of eye movements which are as long as the epochs used for scoring sleep stages turns an 'eye movement profusion' score into an index of how much of the REM is 'active' and how much is 'quiescent'.

Hauri and Hawkins (1971) briefly discuss the various measures which have been used, although they unfortunately refer to EM "density" where they should use "profusion". However, they do recognise that the short and long epochs have different meanings. They chose to use the number of 2.5 epochs with EMs as an "EM index", and the number of 30 sec epochs with EMs as an indicator of "the percentage of phasic REM".

5. Reliability of sleep stage scoring:

Despite large between-subject differences in the characteristics of a night's sleep, Williams et al (1964b) reported that the sleep of a given individual is highly consistent from night to night. Thus an individual who spends 22% of one night's sleep in REM sleep is likely to spend 22% of the next night's sleep in REM too. One exception to this is the so-called 'first night effect' (Agnew, Webb and Williams, 1966). The first night of laboratory sleep - presumably the first night in any strange environment - is characterised by more wakings, more shifts between stages, and longer latencies to stage 4 and to REM sleep, with slightly lower amounts of REM sleep during the night. The mean latency to REM sleep after sleep onset (stage 2) was 106 min on the first night, but dropped to 85 min by the second night in the study by Agnew, Webb and Williams.

Webb and Agnew (1969) examined the correlations between the amounts of each stage on successive nights after the first night, and found good correlations for NREM sleep stages - between .5 and .7 - on successive nights. Correlations for REM sleep on successive nights were somewhat lower - just under .5 for the 20-30 age group. More recently, Moses et al (1972) have examined correlations over successive nights for 20 subjects aged 17 to 21. They found significant correlations (.4 to .6) across nights for the percent time in each NREM sleep stage, but no significant correlation for REM sleep across nights, nor for the delay to sleep or delay to REM sleep, nor for total sleep time (TST). It is in fact not surprising that if TST is not reliable across/

/across nights, then percent REM sleep should not be either, for while the amount of slow-wave sleep is relatively independent of total sleep time (Hartmann et al 1971b), not only does the absolute amount of REM sleep increase with increasing TST, but so does the percentage of REM sleep (Lewis, 1969a). We do not know whether REM sleep amounts would be reliable across nights were TST controlled.

Reliability across nights essentially gives an idea of how representative is one night's sleep rather than another, and needs to be borne in mind if we can only sample one night of sleep. What of the reliability of sleep variables between scorers? Monroe (1969) reported an extensive investigation of reliability between 28 raters in 14 laboratories. One man's record for one night was scored by all. Variation in the amounts of NREM sleep scored was large, the standard deviation (s.d) being roughly one third of the mean score for stages 1, 3 and 4. The s.d. for stage 2 was only one tenth of the mean time scored, while for REM sleep the s.d. was only one twentieth of the mean amount of REM sleep scored. Experience in scoring records led to a higher consistency between scores for the NREM sleep stages. For scoring REM sleep, total sleep time, delay to sleep and awakenings, however, greater experience was not a factor in inducing greater consistency. In fact it emerged that TST, delay to sleep onset, and the amount of REM sleep could be scored with relatively high reliability between scorers.

At about the time of Monroe's paper, a standard manual was published (Rechtschaffen and Kales, 1968). Vogel et al (1972) have studied reliability of sleep stage scoring (within one/

/one laboratory) since the standard scoring system has been issued, and reported percentage agreements on the number of 30 sec epochs scored in any one stage. They found agreement was below 90% for waking, drowsiness, and stages 3 and 4. For stage 2, REM sleep and TST it was above 95%. These reliabilities had, however, been increased by two changes in the standard criteria designed to increase reliability. Nevertheless, it is clear that the discrimination of the various NREM sleep stages by different scorers is not a reliable process, but the scoring of REM sleep is a highly reliable process in the normal subject.

The use of drugs (in the second of these studies) poses fresh problems. Because drugs which affect sleep may also affect the EEG, reliability of sleep stage classification may be affected. Both barbiturates and benzodiazepines (nitrazepam, chlordiazepoxide, diazepam) may introduce fast activity to the EEG in drowsiness and REM sleep, as in Figs. 16 and 17. Moreover on high doses, these drugs may cause a little confusion. For instance, in the transition between stage 2 and REM sleep, 12-14 Hz activity (spindles) in stage 2 may become faster in frequency until an 18Hz activity is present with occasional eye movements. The transition between REM and NREM sleep is then harder to define than in drug-free records. For this reason a retrospective study was made of test-retest reliability in these studies. Five placebo subject-nights (excluding withdrawal) and five subject-nights on drug were rescored for all sleep stages six months after the last record had been scored. The results are given in Table 1.

It can be seen from Table 1 that reliabilities for stage REM, stage 4 and stage 1 are very high. Reliabilities for wakefulness during the night, and for stage 2 are moderate. The reliability for stage 3 is very poor, as a correlation coefficient between two judgements it is only just significant. This very low reliability for stage 3 scoring was also noted by Monroe (1969), and its recognition has no doubt been one reason for the increasing tendency for sleep researchers to combine stages 3 and 4 into one category, "slow-wave sleep".

Also given in Table 1 is the correlation between separate scorings of the same ten records for eye movement profusion. It can be seen that with a correlation of .98 between two judgements this is a highly reliable measure.

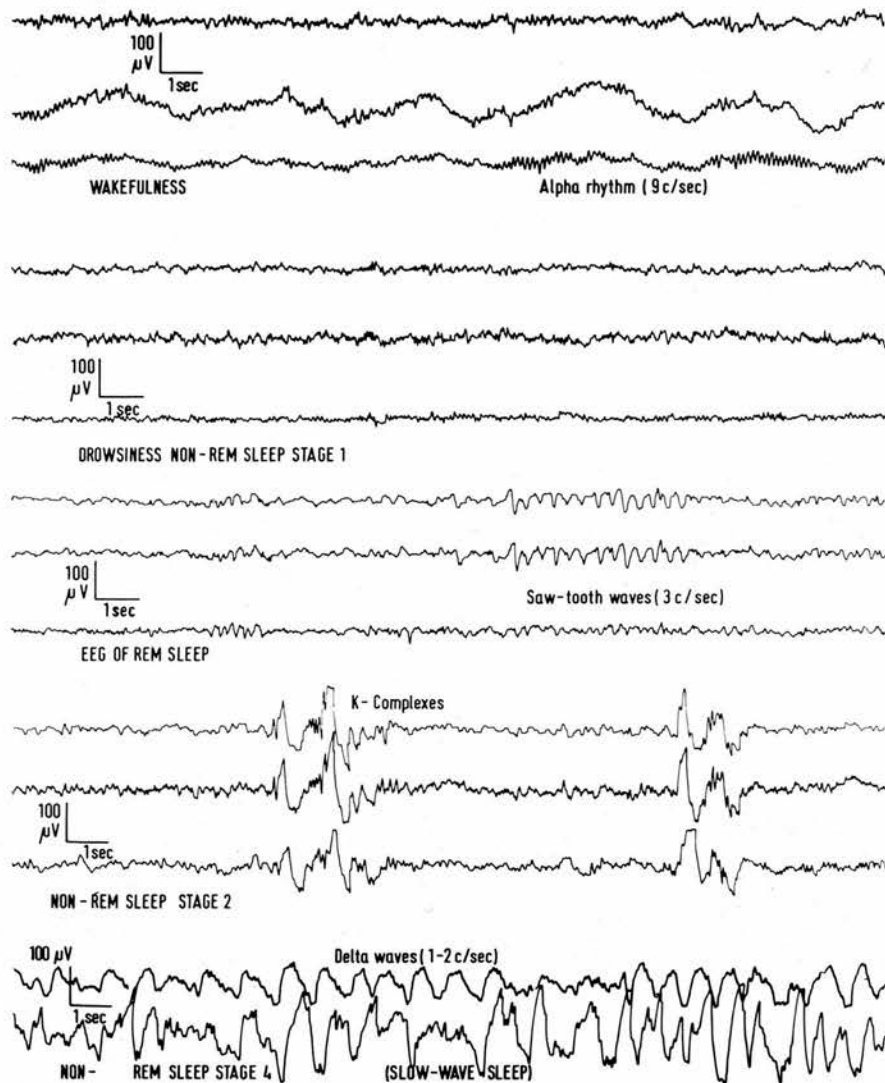


FIGURE 1. THE EEG IN WAKEFULNESS, DROWSINESS, REM AND NON-REM SLEEP. Note the alpha rhythm in wakefulness, the saw-tooth waves in REM sleep, and the K-complexes in stage 2. The upper channel(s) are in each case frontal derivations; the lower channel is a parietal derivation.

TABLE 1.

RELIABILITIES FOR SLEEP STAGE SCORING AND FOR EYE MOVEMENT PROFUSION

Sleep stages:

Test-retest reliabilities for ten records rescored at least six months after they had been scored originally. The ten records comprised five placebo, and five 'drug' records chosen randomly.

Pearson product-moment correlation coefficients:

Wakefulness	: 0.86
Stage 1	: 0.96
Stage 2	: 0.81
Stage 3	: 0.62
Stage 4	: 0.95
Stage REM	: 0.95

Eye movement profusion scores:

Test-retest reliabilities for the same ten records, for the number of two second epochs with eye movements, in the five minutes preceding each experimental REM awakening.

Pearson product-moment correlation coefficient is 0.980

A. HABITUATION IN SLEEP

CHAPTER TWO

LEARNING AND SLEEP

1. Learning and discrimination in sleep:

Habituation is one of the simplest forms of learning: it is learning not to respond to an irrelevant stimulus. Definitions of habituation usually include two elements. First, habituation is seen as a decline in some (unlearned) response as a function of repeated stimulation. Second, many definitions include the stipulation that habituation be a 'central' event, to be distinguished from receptor adaptation or effector fatigue (Harris, 1943; Thompson, 1967). Thompson (1967, p.499) defined habituation as "a decrease in response as the result of repeated stimulation. It is usually distinguished from receptor adaptation (a decrease in receptor response to a constant stimulus), and from effector fatigue ... in other words, habituation is generally believed to be a central event". Martin (1964) has however criticised this emphasis upon habituation as 'central'. She argues "Such views are ineluctably related to a somewhat outmoded reflex-circuit view of central nervous system organisation, whereas current neurophysiological research points consistently towards extensive modification of incoming signals at every level from the receptor upwards" (1964, p39). Martin goes on to argue that "there is clearly a very strong case for insisting that the term "response decrement" replace the more common adaptation and habituation. While Martin's insistence upon a continuum between sensory adaptation, habituation, conditioned response extinction and higher forms of/

/of learning is probably overstated (parallels between habituation and extinction may be gross rather than precise, as Kling and Stevenson have argued, 1970) - nevertheless, it can be useful to look at habituation within the wider context of learning.

Now It would appear that in sleep learning of relatively simple kinds does not take place. While learning of word-pairs has been claimed by a number of workers, these studies have generally been vitiated by the possibility that material was presented when subjects could have been awake for brief periods in the night without the experimenter's knowledge, since EEG criteria of sleep have rarely been used in sleep learning studies (Lewis, 1968a). Carefully controlled studies by Simon and Emmons (1955, 1956) have shown on the other hand that retention of question and answer type material when subjects were in sleep level C or 'deeper' (sleep stages 2, 3 or 4) was effectively zero, or could be accounted for by guessing. Moreover, recognition scores (generally regarded as a more sensitive test of learning than unaided recall) were no higher for subjects exposed to material in sleep levels B to E (stages 1 to 4) than for a control group. A recent study by Bruce et al (1970) also failed to find any evidence for the ability to learn pairs of nonsense syllables during slow wave sleep. Nevertheless in a recent study Evans and Orchard (1969) did show 10% correct recognition of material presented in stage 2 sleep when tested the following morning, using subjects specially selected for responsiveness in sleep who were given a pre-sleep 'set' for sleep/

/sleep learning. These particular aspects of subject selection and induction of a 'set' to learn were chosen deliberately to reproduce current Soviet methods in sleep learning experiments¹.

Unfortunately, at the time of the Simon and Emmons studies it was not customary to distinguish REM sleep from stage 1 (NREM) sleep, and we cannot know which occurred when they refer to 'stage B'. It has been postulated that REM sleep functions to consolidate memory memory, 1965: (Dewan, 1968, 1969) - in contrast to NREM sleep which as Portnoff et al (1966) showed, allows less retention of material previously presented than does a comparable period of wakefulness. Even if consolidation of learning does take place in REM sleep, we have no reason to expect retention of material presented during ongoing REM sleep to be good - indeed we should rather expect the reverse if external stimuli were 'blocked off' in some way to allow consolidation to take place. If we identify Simon and Emmons' stage B with REM sleep, we can conclude that learning is no more possible in REM than in NREM sleep. Recently, however, Hoskovec and Cooper (1969) achieved 30% retention of Russian-English word pairs presented in REM sleep to subjects given posthypnotic suggestion that they would learn the words. This is in contrast to 90% retention in a waking control (without posthypnotic suggestion). The authors concluded that learning in REM sleep was 'possible but not practical'.

¹As I have argued elsewhere (1971) studies such as those by Simon and Emmons do not invalidate the idea that learning while the subject is in bed is possible - here, however, we are concerned solely with the question of whether subjects can learn while actually asleep.

Conditioning is a relatively simple form of learning, almost as elementary as habituation. It would seem from McDonald's (1966) study that conditioning is not possible even in very light sleep. McDonald found no evidence at all of conditioning in a group with an average sleep rating of 1 (on a scale of 0-4, 0 being alert wakefulness). McDonald actually classified in his light sleep group all who "showed EEG evidence of sleep" at any point in the hour long experiment. However, Beh and Barratt (1965) and Weinberg (1966) have both reported some success, Beh and Barratt in particular managed to condition and extinguish the EEG K-complex - a stage 2 response to a tone stimulus - using a mild shock as the unconditioned stimulus (this did not waken the subjects and produced only K-complexes). The conditional stimulus was a 300 HZ tone; a 500 HZ tone not paired to shock served as a control. Weinberg required a motor response from his subjects, so the influence of arousal shortly after the stimulus cannot be ruled out. Beh and Barratt on the other hand do seem to have demonstrated conditioning during ongoing sleep. I believe however that their results are in fact consistent with McDonald's, since he tried conditioning in what would generally be regarded as drowsiness (EEG stage 1, presumably to be identified with McDonald's rating 1). Now considering together the results of McDonald's et al (1964) - they found heart rate response habituation was not possible in drowsiness - and those of McDonald and Carpenter (1966) - they found heart rate response habituation was possible in sleep proper - it seems that simple learning processes may be especially difficult in drowsiness or very light sleep. So it/

/it may well be that conditioning is in fact possible in any but the lightest sleep.

Oswald (1959, 1960), studying the way in which repetitive stimulation (in this case electric shocks) led to the development of sleep, reported habituation of the skin potential response in subjects as they developed EEG signs of drowsiness. Both Pavlov (1927, p256) and Sokolov (1963a, p120) report that the development of drowsiness coincides with habituation of the OR.¹ Bohlin (1971), specifically investigating the interrelationship between sleep onset and habituation, found a highly significant correlation between the two. However, although it would appear that the development of drowsiness and sleep is associated with the extinction of electrodermal responses initially present in the alert subject, it does not follow that drowsiness is an inhibitory condition which prevents responses that might occur otherwise in sleep or wakefulness (Karimova, 1961). For if stimuli are continued, responses may reappear, and they are then very persistent (Sokolov, 1963a, p120, 121; Sokolov and Paramonova, 1961).

Habituation is simpler than conditioning in that it does not require the formation of a connection between two stimuli, being merely a response decrement due to repetition of a stimulus where the response to the stimulus is unlearned. But habituation requires that at some level of the nervous system a 'memory' must operate over the time interval between successive stimuli, as Stern (1968) has pointed out, since the essence of habituation is that the/

¹Orienting, reaction, response, reflex.

/the response to the second and subsequent stimuli is not the same as the response to the novel presentation of the stimulus.

Following a model of the habituation process along the lines of Sokolov (1960), the mechanisms which will be necessary for habituation to occur in sleep are as follows. First, the stimulus must be able to pass any filters in order to be registered. The test of this is simply whether or not a stimulus produces an observable response. Second, the organism must be able to store a 'model' of a stimulus for at least the interval between successive stimuli (the interstimulus interval). Third, the organism must be able to discriminate the trace or model of the initial stimulus from the following stimulus sufficiently well to determine a 'match' or a 'mismatch' between the two.

Can humans discriminate while asleep? That complex discrimination of meaningful information is possible in NREM sleep has been demonstrated conclusively by Oswald et al. (1960). They showed that during stage 2 sleep subjects discriminate between their own and other names. Oswald et al presented subjects' own names and 55 other names under identical conditions, and using blind scoring, showed that EEG responses occurred significantly more often to 'own' names than to others' ($p < .001$). Moreover, this discrimination must have occurred before the EEG arousal response, since it was the occurrence of these K-complexes which discriminated between the names. Further, this effect was significant ($p < .01$) when all trials on which the subject showed movement or arousal to a lighter stage of sleep were excluded, demonstrating that this discrimination could occur in ongoing sleep. Subjects were used as controls for each other since /

/since among the 56 names used were the names of all staff and students in the laboratory, and the recording was made up before it was known which of these would be the subjects. Even had the names of some laboratory members been spoken louder, the technique of using one recording for all subjects controlled for the effect of extra stress leading to increased responding to the subject's own name. That this discrimination is not the result of some overlearned mechanism for responding to one's own name was shown also. Subjects were instructed to respond by awakening and fist clenching to one name other than their own before they went to sleep. Significantly more hand movements were shown to this other name than to any other of the 54 names ($p < .001$). Lastly, it was also shown that subjects could discriminate between names and meaningless sounds - the names played backwards - during sleep.

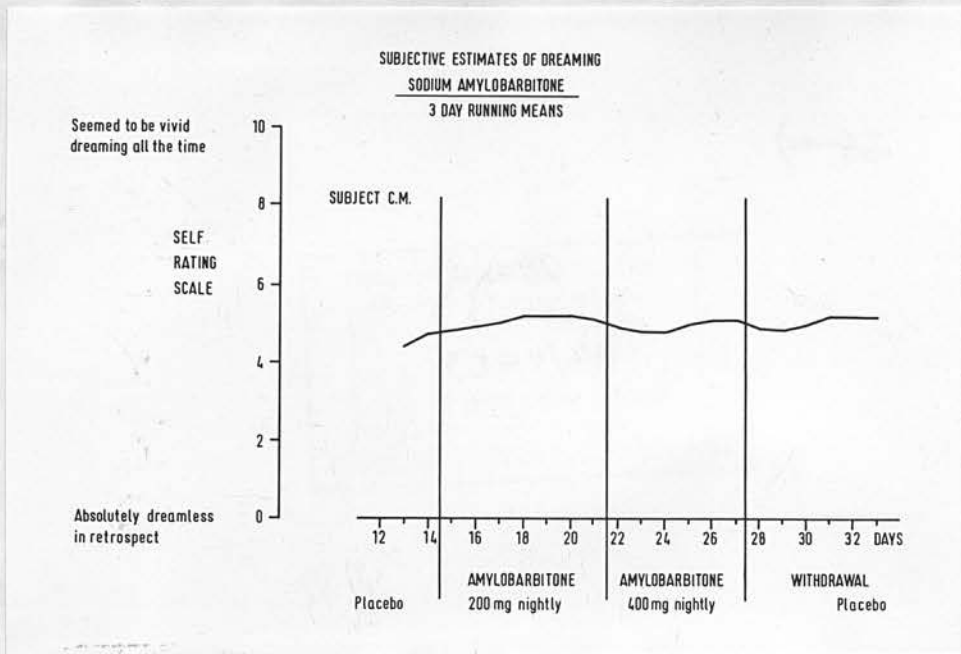
Discrimination, as evidenced by differential incorporation of meaningful material, is also possible in REM sleep. Castaldo and Holtzman (1967) showed that playing a subject's own voice during REM sleep resulted in dreams in which the subject was more active, assertive and independent than if some other person's voice was used. Berger (1963) using blind matching, obtained a highly significant correspondence between names played to subjects in REM sleep and the content of their dreams reported on subsequent awakening. Dream incorporation of names in distorted fashion shows that in REM sleep the subject not only processes the stimulus as a name rather/

/rather than a meaningless sound, but can register and process the content of that name; further, this information must be 'stored' for the time between the stimulus and the subsequent awakening when the dream narrative is delivered. Discrimination of stimuli in REM sleep is evidenced also by the different arousal threshold for signal and non-signal stimuli in REM sleep (Williams et al 1966).

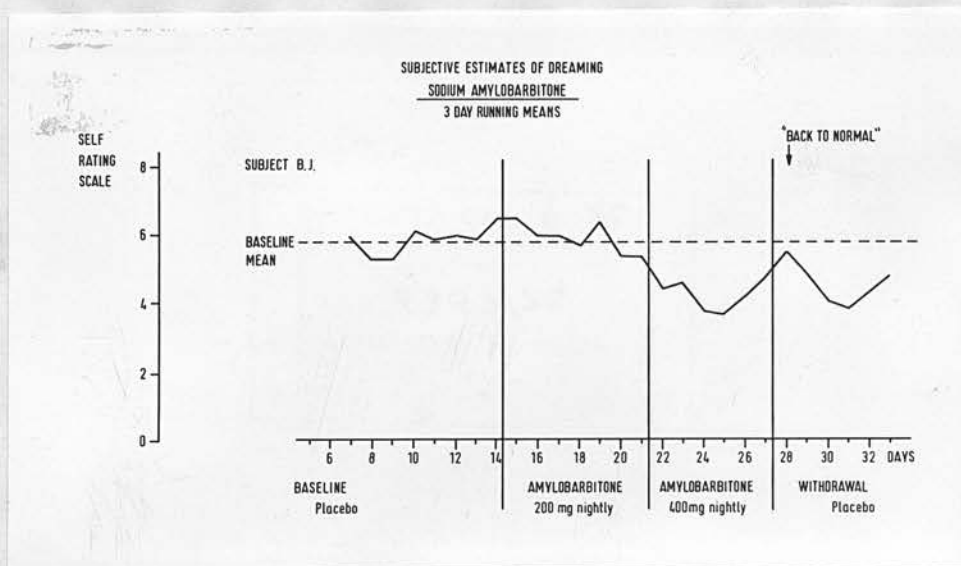
In REM sleep both discriminative ability and storage of externally presented stimuli can occur. These abilities imply that habituation in REM sleep might at least be possible. (Not that there need be any connection between the short term 'memory' processes underlying habituation, and processes underlying short term memory of the sort that would be required for dream recall, for instance). In NREM sleep discrimination can occur, but there is little to suggest functioning memory processes. Besides the evidence that 'sleep learning' itself is hardly possible in NREM sleep, it has been argued that a failure of memory in NREM sleep is the chief reason for less recall of content after NREM wakings than after REM wakings (Berger, 1971). However, in so much as habituation may involve different 'memory' systems from those employed in sleep learning, we might still expect to find that habituation is possible in sleep.

2. Habituation and Sleep: Neurophysiology tells us nothing:

Reviews of work on habituation from a physiological standpoint include Thompson and Spencer (1966), Thompson (1967), Hinde (1966) and Horn and Hinde (1970). The psychologist, rather than/



(a)



(b)

FIGURE 28. SUBJECTIVE ESTIMATES OF DREAMING: TWO SUBJECTS ON AMYLOBARBITONE.

- (a) Subject C.M. experiences no change in dreaming during the course of the experiment.
- (b) Subject B.J. experiences less dreaming while on the full dose of amylobarbitone. He reports his dreaming is back to normal on withdrawal of the drug.

/than physiological interest here is upon what sorts of mechanisms might be responsible for habituation rather than the exact nature of those mechanisms (Deutsch, 1960, p.10-12).

The model of the orienting reaction (OR) and its habituation Sokolov put forward in 1960 demands an inhibitory effect by the cortex on the reticular formation (r.f.) during habituation. Now it does seem likely that the reticular system, which mediates behavioural arousal, mediates behavioural and autonomic aspects of the OR (Sharpless and Jasper, 1956). Moreover, that the cortex has inhibitory effects on the reticular formation has frequently been argued (Dell, Bonvallet and Hugelin, 1961; Jouvet, 1961). Such observations as those on rabbits by Horn (1966), in which he has shown that habituation of single cell responses to sensory stimulation can be blocked by removal of the neocortex, do support a model of Sokolov's type, with active inhibition of the r.f. by the cortex, the crucial step in producing habituation.

Ablation of structures however is not necessarily a reliable method of determining their function (Deutsch, 1960, p.167-170). While it seems clear from such studies that the cortex plays some role in the development of habituation, it is difficult to predict the effect of sleep. Despite Mackworth's statement (1969, p101) that the (supposed) lack of habituation in sleep might be due to the removal of cortical inhibition, the cortex is not ablated in sleep! There is no direct evidence on whether NREM sleep affects 'cortical inhibition' in a way which could interfere with habituation.

That cortical activity might be depressed in either NREM or REM sleep (and Evarts findings (1961) give us no reason to suppose this) does not itself imply that any specific cortical inhibitory action mediating habituation is itself necessarily depressed or abolished.

Thus Mackworth's statement is too vague to be of use. If Douglas and Pribram's (1966) theory of learning is anything to go by, to predict the effect of sleep on habituation we should need to have far more precise information on changes in the activity of specific inhibitory neurons from wakefulness to sleep. Douglas and Pribram's theory involves the hippocampus and frontal cortex in a complex interplay of 'collateral' and 'recurrent' inhibition. Collateral inhibition here is the inhibition of one neuron by another; recurrent inhibition is self inhibition by a single neuron due to its own activity; these two mechanisms are seen as being mutually antagonistic. Pribram's (1967) model of the response to a repetitive stimulus involves:

- (1) an alerting reaction or orienting reaction, a function of 'enhanced contrast' produced by collateral inhibition;
- (2) focussing and registration, mediated by the frontotemporal cortex; and
- (3) habituation, due to an enhancement of recurrent inhibition.

It is clearly possible on Douglas and Pribram's model that NREM sleep, if it were associated with changes in the activity of inhibitory neurons in the cortex, would affect habituation; but it is impossible/

/impossible to tell in which direction. So slight a change in alertness as asking subjects to "pay attention" can abolish habituation (Korn and Moyer, 1968) - what on earth will sleep do? Neither Sokolov's simple model of 1960 nor Douglas and Pribram's theory are much help; we simply do not know enough either about which structures are important in habituation nor about what sleep does to neuronal activity to predict what sleep will do to habituation.

CHAPTER THREE

THE "ORIENTING REACTION"

1. The "Orienting Reaction" in Wakefulness

The "orienting reflex", "response" or "reaction"¹ (OR) as it is variously called, occurs to a novel stimulus, and typically involves many components, postural, autonomic, and electroencephalographic (Sokolov, 1960, 1963a, b; Lynn, 1966; Johnson and Lubin, 1967). I shall concern myself here with responses to auditory stimuli, and with the autonomic and EEG response components, and not attempt to review all the literature on the OR. In an awake animal, provided the stimulus is not too intense, painful, or conditioned to another stimulus, the responses will normally habituate if the stimulus is repeatedly presented. (However, Furedy (1968, 1969) has reported that plethysmographic changes do not necessarily habituate, which poses problems for a conventional view of the OR: he warns against assuming that all autonomic responses are necessarily part of the OR).

The literature on the OR is extensive, and much of it has come from the Soviet Union and from Sokolov in particular. Razran (1961), Gray (1966) and Lynn (1966) have provided reviews of the Soviet literature. A good deal of this, and many of the studies in the West, concern the relationship between the OR and conditioned responses. However, in this study the components of the OR have been used to determine whether habituation of an unlearned response can occur in states of sleep.

¹ I have chosen the term orienting reaction rather than response or reflex in this study because the phenomenon manifests itself as a complex of several responses in different modalities: it is not one response or reflex at all, for the different components do not always occur together even in the awake alert organism. Where other terms are used, it is either because only one component of the reaction is being referred to (i.e. one response), or when the terminology used by the original authors has been retained.

Thompson and Spencer (1966) set out what they saw as the chief 'laws' of habituation, namely a decrease in response to continued stimulation, spontaneous recovery, generalisation and dishabituation. Thompson and Spencer also reviewed psychological contributions to the theory of habituation. McDaniel and White (1966) have tried to take this review of theory to the point of making predictions which can discriminate between the theoretical approaches. They see three main viewpoints: 'exhaustive' theories (Skinnerian, reinforcement being the central concept), 'inhibitory' theories deriving from Pavlov (1927) and developed by Eysenck (1963), where habituation is seen as a process essentially comparable to extinction, and 'anticipatory' theories (Sharpless and Jasper, 1956) - Sokolov's model is essentially of this sort. McDaniel and White seek to discriminate the inhibitory theories, from the anticipatory by their predictions as to the effects of random versus regular intervals between stimuli. Unfortunately their predictions would appear to rest upon the assumption that the 'reactive inhibition' (Pavlov's "internal" inhibition) resultant from a response decays linearly with time. If it did, then they would be correct in saying that reactive inhibition theories would predict no difference in rate of habituation between regular and irregular interstimulus intervals. If the reactive inhibition which accumulates to produce habituation does not decay linearly with time, however, then McDaniel and White's "test" of the theory breaks down. It is in fact noteworthy that models of habituation have been almost incapable of producing testable/

/testable hypotheses. All three approaches mentioned above allow no predictions to be made with confidence about the effect of sleep on habituation.

Predictions from a reactive inhibition model rely too heavily upon the supposed parallel between extinction and habituation; this parallel has been shown to have defects - essentially the comparison is too gross (Kling and Stevenson, 1970). If it is possible to make any specific prediction about habituation in sleep from Eysenck's ideas, it is that since depressant drugs increase reactive inhibition, and induce sleep, then sleep itself should, by increasing reactive inhibition, lead to more ready habituation than is found in waking.

Sokolov's model of the OR habituation process (1960) involves two hypothetical systems, a system for the formation of a model of the stimulus, and an amplifying system. The system for model formation is supposed to be cortical, and the amplifying system is supposedly the ascending reticular activating system. Stimulus information is fed both to the amplifying and the model formation systems. The output from the amplifying system provokes an OR, unless inhibited by the modelling system. This only occurs after several stimuli have enabled the latter to build up a 'model' or 'trace' of the stimulus; then, if an incoming stimulus is 'matched' with a stimulus model, the match-mismatch model system sends inhibitory signals to the amplifying system and thus prevents the occurrence of an OR. Additionally, there is provision for the amplifying/

/amplifying system to inhibit the match-mismatch system - this is supposed to occur for instance when a stimulus in sleep causes an arousal reaction.

It is supposed that as drowsiness and sleep develop, cortical activity is inhibited, and so the (cortical) match-mismatch system is unable to inhibit the OR. This explains why the OR is so difficult to extinguish in drowsiness (Sokolov, 1963a, p.120). It might then be hypothesized that in 'deep' sleep, NREM stages 3 and 4, where there is supposedly a high level of inhibition of cortical activity, cortical inhibition of the OR will be at a minimum. Habituation might thus be hypothesized on Sokolov's model to take longer in stage 4 than in stage 2, and longer in stage 2 than in REM sleep. Sokolov follows Pavlov (1927) in linking the development of sleep with the development of "cortical inhibition" (inhibition of cortical activity).

Jouvet and Michel's finding (1959) that habituation of the OR was not possible in the decorticate cat is consistent with this hypothesis; also, Kleitman and Camille (1932) did not find habituation possible in decorticate dogs while they were awake.

Predictions made from Eysenck's concepts and from Sokolov's thus are at odds. Eysenck's concept of inhibition appears a trifle simplified by comparison with Sokolov's, but perhaps this is not fair as Eysenck has never himself discussed the role of sleep on conditioning and extinction processes. However, in the light of Evarts' work on single unit activity (1961) it might seem unnecessary to suppose that cortical inhibitory influence on Sokolov's r.f. amplifying system would be in any way depressed during sleep.

Sokolov (1963a) distinguished an orienting reflex (OR) from a defensive reflex (DR) and an adaptive reflex. The adaptive reflex is specific to the modality of the stimulus, and to the sense or direction of change of the stimulus (for example, peripheral vasodilation or vasoconstriction to heat or cold). Both DRs and ORs are non-specific to the modality of the stimulus, and non-specific to the sense of stimulus change. The DR is normally shown to painful or very intense stimuli; the OR to novel stimuli which are not noxious.

Sokolov (1963a) distinguished the OR and the DR on the basis of several criteria. An OR is elicited by low to moderate intensity stimuli, a DR by intense stimuli. Secondly, it is assumed an OR will habituate rapidly in an awake organism if the stimulus is repeated; the DR will habituate slowly or not at all. Thirdly, Sokolov maintains that an OR is accompanied by cephalic vasodilation and a DR by cephalic vasoconstriction - however, Raskin et al (1969) argue that cephalic vasomotor responses do not discriminate ORs and DRs.

These criteria are of two kinds: criteria of the stimulus, or 'definitions', and criteria of the form and subsequent behaviour of the response. Now the object of the present study is to see whether a process which occurs readily in the wakeful organism - habituation - may be altered or blocked during sleep. The form and components of a response elicited in sleep differ from the form of the wakeful OR: that heart rate responses (HRRs) to equal/

/equal intensity tones are different in form in sleep and wakefulness has been shown by Hord et al (1966). Thus the criteria defining an OR in terms of response characteristics are not necessarily reliable when translated from wakefulness to sleep, particularly since there is no general agreement about what these criteria are. Furthermore, Berg et al (1971) suggest that the threshold for DRs may be lowered during sleep. While Graham and Jackson (1970) argue on the basis of cephalic vasomotor responses that a stimulus eliciting an OR in wakefulness elicits a DR in sleep, Raskin et al (1969) argue that such responses do not discriminate the two reactions even in wakefulness. Because of this confusion, it seems necessary to compare habituation in sleep and wakefulness using identical stimuli - and to determine whether the response itself is an OR or DR or a startle response, by looking at the wakeful response. I shall therefore turn to a discussion of the characteristics of the responses comprising the wakeful OR. It should, however, be borne in mind that these various responses do not necessarily always occur together as components of "one OR".

Heart rate responses (HRRs) in waking

The literature on heart rate changes in waking subjects following brief auditory stimuli is contradictory; some studies have reported only monophasic changes, while others have reported diphasic responses, an initial heart rate acceleration and a subsequent deceleration. Graham and Clifton (1966) provided an excellent review of studies up to that date. They report a number of studies, including their/

/their own, with stimuli up to 70 db, which found only heart rate deceleration following the tone stimulus. Since Graham and Clifton's review, Headrick and Graham (1969), and also Keefe et al (1971) have reported finding that the only significant response to tones of 60db or less was heart rate deceleration.

Graham and Clifton concluded from their review that stimuli of 70 db or more led to a diphasic (acceleration then deceleration) response. One of these studies was that of Lang and Hnatow (1962). These authors also investigated various possible measures of the diphasic heart rate response, and they concluded that a peak-through measure was most sensitive to the effects of stimulus repetition. Thus it is to be preferred in studies of the habituation of diphasic heart rate responses.

However, Uno and Grings (1965) show results in which a diphasic response is evident to tones as low as 60 db (although in their text they only describe monophasic responses with no relation to stimulus intensity: deceleration to 70-90 db, acceleration to 60 and 100db). More recently Germana and Klein (1968) reported a diphasic response to all intensities between 50 and 90 db.

Meyers and Gullickson (1967) and Meyers (1969) recently found responses which consisted on the first trial of deceleration, but on second and subsequent trials of a diphasic response. They interpreted their findings as an OR (deceleratory response), whose habituation was partially masked by the development of an adaptation reaction. In the first experiment, this acceleratory component/

/component (adaptation reaction) was found to be non-habituating, but the second experiment (Meyers 1969) contradicted this, habituation being complete within 10 trials. Smith and Strawbridge (1968) also found evidence to suggest the development of an adaptation reaction (heart rate acceleration) after the initial trials had elicited a solely deceleratory response to a 40 db tone. Sokolov (1963a) has also mentioned the possibility of adaptation reactions developing as the orienting reaction is extinguished.

Working with dogs, Soltysik et al (1961) reported that the cardiac response to tone-on was acceleration, and that to tone-off deceleration; the response to a brief tone was diphasic. They concluded that these responses were adaptation reactions to auditory stimuli. There are differences between Soltysik's results and those of Meyers and Gullickson and Smith and Strawbridge. Soltysik's "adaptation reactions" habituated (on Sokolov's theory they should not), whereas the "adaptation reactions" of Meyers and Gullickson and Smith and Strawbridge developed as the trials were repeated. Smith and Strawbridge found solely acceleration from the first trial using a brief tone; they asked their subjects to count the tones ("to ensure attentiveness"). All these results are confusing and contradictory, but they do suggest that Graham and Clifton are wrong in supposing acceleration is elicited only as a DR, and that acceleratory responses may be part of an adaptation reaction to tones.

Graham and Jackson (1970) examined the apparent contradictions in the literature and proposed that these could be resolved by/

/by supposing the deceleratory component reflects the 'true' OR, the acceleratory component arising either from a 'startle' reaction (a function of sudden stimulus onset, when rise times are only a few milli sec), or from defensive reactions to intense tones. Graham and Jackson reported that even a 90 db tone elicited merely heart rate deceleration if the rise time was long (300 milli sec). Berg et al (1971) report that the effect of short rise time interacts with state - at least in infants - thus in non-alert subjects, short rise times were more especially likely to produce heart rate acceleration. Hatton et al (1970) point out that startle reactions may also be produced by stimulus onset transients as well as by short rise times.

Thus it would seem likely that high intensity tones (90 db or over) will elicit what Sokolov (1963a) has termed defensive reactions with acceleratory and deceleratory components. Lower intensity tones will elicit responses which include deceleration, and may or may not include acceleration as part of a startle response, or as part of an adaptation reaction as Soltysik and Meyers and Gullickson, have suggested (1961, 1967).

Lacey (1964, 1967) has argued for a distinction between two arousal reactions: those of 'stimulus intake' and those of 'stimulus rejection', which may roughly be identified with Sokolov's orienting and defence reactions. Sokolov does not discuss cardiac changes. However, Lacey suggests that heart rate acceleration would actually inhibit EEG and autonomic activation and therefore proposed that/

/that heart rate acceleration should accompany 'stimulus rejection' (Sokolov's defensive reflex). Lacey's position provided the theoretical basis for Graham and Jackson's and Graham and Clifton's conclusions.

However, in a recent experiment designed specifically to test Graham and Clifton's hypothesis, Smith and Strawbridge (1969) used deliberately low intensity (54 db), slow rise time tones, and found a predominately acceleratory response, which had not habituated within ten trials. In this experiment they doubted the hypothesis that their response might be an "adaptive" reaction, and instead adduced evidence to show that this acceleration is in fact a secondary effect of changes in respiration - and is thus normally to be expected as a response in situations where an 'OR' is anticipated. The issue of whether acceleratory responses represent ORs modified by respiratory changes or adaptation reactions remains unresolved.

The Skin Potential Response

The skin potential response (SPR) is one of two electrodermal ('psychogalvanic') responses manifested to novel stimuli. The galvanic skin response (GSR) is a skin resistance change, whereas the SPR is a change in potential¹. The origin of skin potential fluctuations would appear to be a complex sum of potentials arising in the epidermis and a potential generated by presecretory activity of the sweat glands.

¹The term GSR is sometimes used loosely to refer to any electrodermal response; to avoid ambiguity I shall use the term to refer exclusively to skin resistance responses.

Skin resistance changes also seem to be consequent on changes in electrical activity both in the sweat glands and in the epidermis. Resistance and potential changes cannot however be directly related since they do not always occur together (Wilcott, 1964; Venables, 1967; Hori et al 1970).

Both SPRs and GSRs are widely accepted as components of the OR. However, it is important to note that electrodermal fluctuations are not necessarily indicative of an evoked response to stimulation. Spontaneous electrodermal fluctuations are especially common in stage 4 sleep, when they may occur almost continuously as 'GSR storms' for extensive periods of the night (Oswald et al 1959; Johnson and Lubin, 1966; Lester et al 1967).

Electroencephalographic responses

The electroencephalographic (EEG) response to a novel stimulus is an arousal, non-specific to the stimulus modality. It consists, for instance, in the blocking of alpha rhythm in alert subjects, or the appearance of alpha rhythm bursts in drowsy subjects. The alpha blocking response in waking subjects typically habituates readily (Sokolov, 1960; 1963; Tizard, 1968), although localised aspects of the EEG response do not necessarily habituate (for example occipital alpha blocking will persist if the stimulus is visual).

2. The "Orienting Reaction" in Sleep:

What happens to the "OR" as sleep develops? Sokolov (1963a, p120) states that "the development of drowsiness coincides with the extinction of the orienting reaction". He goes on to say that/

/that "further chronic extinction ... leads to the re-appearance of the orienting reaction ... the reactions so established do not undergo extinction in spite of repetitive stimulation ...". It is not clear whether he specifically refers to all components of the OR. McDonald et al (1964) looked at habituation in both alert and drowsy subjects (their drowsy subjects appear to have been in EEG stage 1). They reported that cardiovascular measures (heart rate and vasomotor components) did not habituate - but the CSR did.

Habituation of the OR may be accompanied by the development of sleep - indeed strong rhythmic sensory stimulation, even electric shocks, may facilitate the onset of sleep (Oswald, 1959, 1960). If light sleep ensues, not just drowsiness, all components of the OR return (Sokolov and Paramonova, 1961; Hord et al 1966; Johnson and Lubin, 1967) except possibly the skin resistance response, which according to McDonald and Carpenter (1966) does not return in sleep.

Thus while repetitive stimulation may sometimes lead to extinction of the OR, and then to sleep, various components of the OR will then return with sleep itself.

Which components of the OR are elicited during sleep by a single (initial) stimulus? Body movements (evidenced by muscle activity on EMG, EKG or EEG records) may occur to high intensity stimuli; these usually accompany arousal either to wakefulness or a 'lighter' stage of sleep. Body movements should therefore not be considered as components of the OR; rather they should be considered part of/

/of the arousal reaction from sleep, of the habituation of which Sharpless and Jasper (1956) made a classic study¹.

EEG responses in sleep:

Phasic responses such as the EEG K-complex need to be carefully distinguished from the more prolonged arousal which occurs to high intensity stimuli. Phasic EEG responses are quite different in REM and NREM sleep. The K-complex may be evoked in stage 2 (NREM) sleep by auditory stimuli at 25 db below the arousal threshold (Keefe et al 1971); it is a response consisting of one or more slow waves of medium to high voltage, often with superimposed 12 HZ activity. In 'deep' slow wave sleep (SWS) (NREM stages 3 and 4) any EEG responses are masked by the high voltage delta waves of SWS, and it is difficult to distinguish a response without averaging techniques.

In REM sleep, besides a very small amplitude evoked response, bursts of alpha activity are observed (see Fig. 2), and slight reduction in voltage and acceleration in EEG frequency are also sometimes detectable.

Cardiac responses in sleep

Cardiac responses seem to be readily elicited in sleep. Thus the HRR to auditory stimuli is definitely present in all stages of sleep, although its magnitude differs between stages. The HRR appears unusual among responses in that its magnitude is greatest/

¹In waking, the OR, DR and startle response are all often considered as "arousal reactions". However, in sleep it is useful to restrict the term arousal reaction to responses which involve change of sleep stage, waking, or EEG evidence of arousal" (K-complex, appearance of alpha).

/greatest in NREM stage 2 and in REM sleep (Hord et al, 1966).

Baust and Marbaise (1971) reported that in REM sleep the accelerative component of the HRR was of about the same magnitude as that in stage 4. But it would appear from their figures that the magnitude of the diphasic response as a whole is largest in REM sleep. In REM sleep the HRR can be elicited at 20 db below arousal threshold, whereas in NREM sleep it can only be elicited by tones 5 db below arousal threshold, according to Keefe et al (1971).

The form of the HRR would appear not to be identical in sleep and waking: the waking response is of smaller magnitude and may consist only of deceleration, as described earlier. Responses in REM sleep, and in stages 2, 3 and 4 are of the same form and latency, and are always diphasic (Hord et al 1966). Hord et al say that the form of the HRR may be the same in sleep and waking, but their graphs do not suggest this; Keefe et al (1971) found essentially the same responses as Hord et al and concluded that there were real differences between sleep and waking in the form of the response. Certainly, Berg et al (1971) found different responses in alert and non-alert infants.

Electrodermal responses in sleep

Electrodermal responses seem in some ways to be complementary to cardiovascular responses in sleep. Higher levels of both evoked and spontaneous skin resistance responses (GSRs) occur in SWS than in REM sleep, whereas heart rate evoked and spontaneous response magnitudes are lower in SWS. In REM sleep where heart rate responses and variability are high, and thresholds for responses low, GSRs/

/GSRs and skin potential responses (SPRs) are least frequent (Johnson and Lubin, 1966; McDonald, Shallenberger, and Carpenter, 1967; Johnson, 1970; Keefe et al 1971).

There are however contradictions in the literature as to whether evoked skin potential and skin resistance responses can actually occur at all during sleep. Broughton et al (1965) and Tizard (1966b, 1968) both reported the occurrence of evoked SPRs to stimuli in sleep; neither examined GSRs. Sokolov and Paramonova (1961) found GSRs in sleep; Johnson and Lubin (1967) reported both SPRs and GSRs in all stages of sleep, REM and NREM. However, Oswald (1962, p34) reported that SPRs vanished with the onset of drowsiness and sleep. SPRs were then rare, but they returned as sleep continued and 'deepened' to stages 3 and 4. Ackner and Pampiglione (1955) also reported that GSRs could not be elicited in "light sleep". Although they observed SPRs, McDonald and Carpenter (1966) reported that evoked GSRs did not occur at all in any stage of sleep. Keefe et al (1971) go so far as to report that neither SPRs nor GSRs occur in any stage of sleep unless the stimuli are loud enough to wake the subject.

These disparate and confusing reports can be understood though, I think. 'Early' stage 2 sleep, shortly following sleep onset, may be a special case. Ackner and Pampiglione may well have examined such early stage 2. Oswald (1962) reported that responsiveness returned within about 30 minutes, so a lack of response might easily have been missed by those workers who were interested in the whole/

/whole night, especially in the process of averaging responses in the manner of Johnson and Lubin (1967). Tizard (1965) allowed about an hour of sleep before commencing her experiment, and so would not have encountered the early sleep effect. It is particularly McDonald and Carpenter's and Keefe et al's report that seem quite at variance with the other literature.

However, inspection of the figures of Keefe et al indicates that they did get 0.1 mV responses at 10 db below threshold in REM sleep and in SWS; only in stage 2 did they not. Lastly, they calculated response magnitudes by subtracting the 'pseudo-responses' to 'pseudostimuli' marked 25 sec before the actual tone from the evoked responses. Since they allowed a very long criterion for latency (0-10 sec, in contrast to the more usual 1.5 - 3.5 sec used by Tizard), they were highly likely to score a 'pseudoresponse' from spontaneous activity. Subtracting these 'pseudoresponses' from the actual responses (their method of controlling for spontaneous variability) must further have reduced their chances of detecting responses. The technique of subtracting each 'response' to each 'pseudostimulus' from the real response will always reduce the magnitude and increase the variance of measured responses beyond their 'true' values, and as such is a poor procedure for taking spontaneous variability into account.

3. The recording of electrodermal and cardiac measures of the "orienting reaction", and physiological factors controlling these measures:

(a) Electrodermal responses

Vigouroux, in 1879¹, was the first to report electrical responses of the skin in response to external stimuli. Both Féré², in 1888, and Tarchanoff³, in 1890, studied the phenomenon shortly after. Féré described the skin resistance response (SRR) under conditions when an external current was applied to the skin; Tarchanoff studied the natural potential differences between differing points on the skin; hence the skin resistance response and the skin potential response (SPR) have come to bear the names of Féré and Tarchanoff respectively.

C.W. Darrow in the nineteen twenties and thirties pioneered work on the physiological origins of the SRR and the SPR. Much recent work has altered little the conclusions to which Darrow arrived. Darrow developed a method for continuously recording the moisture liberated from the skin - most of which could be accounted for as due to sweating. Darrow then showed that there was a correlation between sweating and galvanic skin reactions, but he also showed that this was far from the whole picture. He described (1929)⁴ how following a stimulus, the initial negative wave of the SPR appeared with an average latency of 1.2 sec, and the positive component appeared after 1.8 sec on average. The moisture from the sweat glands did not appear until 3 sec had elapsed.

¹Vigouroux, R. (1879) Comptes Rendues Soc. Biol. 31 336.

²Féré, C. (1888) Comptes Rendues Soc. Biol. 40 217.

³Tarchanoff, J. (1890) Pflügers Arch. 46 46.

Darrow adduced his evidence to argue that the decreased resistance of the skin in the SRR was not a function of the presence of conducting moisture on the skin. Darrow suggested that the breakdown of a semi-permeable membrane to ionic flow was crucial to the development of skin potential and resistance responses.

In the same year⁵ Darrow set out to test the idea held by both Vigouroux and Féré that the SRR was mediated by vasomotor mechanisms. Darrow concluded that since SRRs preceded vasoconstrictions on average by about two seconds, it was unreasonable to suppose that vasomotor changes were responsible for the occurrence of SRRs.

Between 1934 and 1937⁶ Darrow set out to examine in greater detail the correlation he had earlier found⁴ between electrodermal reactions and perspiration. He found that perspiration rates were related to resistance changes by a hyperbolic relation, but were related to conductance changes linearly. On the supposition that psychological activity is generally related logarithmically with physiological activity, Darrow recommended the use of log conductance changes as the SRR measure of choice.

⁴Darrow, C.W. (1929) Sensory, secretory and electrical changes in the skin following bodily excitation. J. Exp. Psychol. 10 197-226.

⁵Darrow, C.W. (1929) The galvanic skin reflex and finer-volume changes. Amer. J. Psychol. 88 219-229.

⁶Darrow, C.W. (1934) The significance of the galvanic skin reflex in the light of its relation to quantitative measurements of perspiration. Psychol. Bull. 31 697-698.

Darrow, C.W. (1937) The equation of the galvanic skin reflex curve. J. Gen. Psychol. 16 285-309.

Carmichael et al. (1941)⁷ demonstrated that the SRR was decreased by atropinisation, but not abolished, whereas sweating was abolished under these conditions. Ex-sanguination also reduced the SRR, and a combination of this and atropinisation was capable of abolishing the SRR completely. However, Lader (1970)⁸ showed that atropine takes about 50 min to take full effect, and will then abolish the SRR completely. This implies a cholinergic mechanism for the SRR (i.e. it is mediated sympathetically). The fact that SRRs can be shown in this way to be associated with sweat gland activity has encouraged the development of a model for skin resistance in which sweat glands are seen as resistances in parallel. Rothman (1954)⁹ has in fact developed this model most clearly: using the evidence accumulated by Darrow, he developed a model in which the reduction in resistance is seen as due to the pre-secretory electrical activity of the sweat glands. However, Rothman emphasised that the sweat glands were not solely responsible for electrodermal responses, and he stressed the involvement of both the semi-permeable membranes of the sweat glands, and of an epidermal semi-permeable membrane. Moreover, Wilcott has more recently performed a series of experiments in which he has both confirmed the original work of Darrow, and has fairly conclusively shown that both skin resistance and skin potential responses are partially/

⁷ Carmichael, E.A., Honeyman, W.M., Kolb, L.C. and Stewart, W.K. (1941). A physiological study of the skin resistance response in man. *J. Physiol. (Camb.)* 22 329-337.

⁸ Lader, M.H. (1970) The unit of quantification of the G.S.R. *J. Psychosom. Res.* 14 109-110.

⁹ Rothman, S. (1954) *Physiology and biochemistry of the skin*. University of Chicago Press, Chicago.

/partially but not wholly dependent on the presecretory activity of the sweat glands¹⁰.

In this series of experiments (superceding his earlier experiments, in which he came to other conclusions), Wilcott showed that atropine affected sweating, skin resistance, and skin potential. This clearly indicates that both skin potential and skin resistance are at least partly mediated by a sympathetic mechanism involving cholinergic fibres. But Wilcott also found that skin potential and skin resistance both recovered from the effects of atropine more rapidly than did sweating; it was from this observation that he concluded that skin resistance and potential were partially independent of sweat gland activity. Wilcott also demonstrated that the amplitude of the positive component of the SPR increased as basal skin potential level increased in negativity, and the negative component amplitude decreased under these conditions. Since these relations were both linear and of the same magnitude, it would follow that the amplitude of the diphasic SPR might be largely independent of basal skin potential. This is consistent with Wilcott's earlier finding¹¹ (1958) that SPR amplitude was independent of basal potential level.

¹⁰Wilcott, R.C. (1964) The partial independence of skin potential and skin resistance from sweating. *Psychophysiology* 1 55-66.

¹¹Wilcott, R.C. (1958) Correlation of skin resistance and skin potential. *J. Comp. Physiol. Psychol.* 51 691-696.

Edelberg¹² (1964) has confirmed Wilcott's conclusion that not only SPRs but also SRRs are partially independent of sweat-gland activity. This is the conclusion reached by Martin and Venables' review (1966)¹³. If the skin potential basal level, as well as response, is affected by atropine as Wilcott found¹⁰, then the most conservative conclusion would seem to be that SPRs, SRRs, potential level and resistance level are all partially dependent on the presecretory activity of the sweat glands, and partially dependent on the functioning of an epidermal membrane, though each would seem to be dependent on these mechanisms in differing degree.

Martin and Venables¹³ also discuss central factors affecting electrodermal responses. There is very little work on the central areas involved in their control, with humans, apart from the observation that severance of the spinal cord, or of sympathetic nerves, leads to disappearance of the SPR. There is no clear evidence beyond this, which is not confounded with the effects of lesions in the c.n.s. on skin temperature. Venables (1955), Venables and Sayer (1963) and Edelberg and Burch (1962)¹⁴ have shown that skin temperature can affect electrodermal responses.

¹²Edelberg, R. (1964) Independence of galvanic skin response amplitude and sweat production. *J. Invest. Dermatol.* 42 443-448.

¹³Martin, I. and Venables, P.H. (1966) Mechanisms of palmar skin resistance and skin potential. *Psychol. Bull.* 65 347-357.

¹⁴Venables, P.H. (1955) The relationships between P.G.R. scores and temperature and humidity. *Quart. J. Exp. Psychol.* 7 12-18.

Venables, P.H. and Sayer, E. (1963) On the measurement of the level of skin potential. *Brit. J. Psychol.* 54 251-260.

Edelberg, R. and Burch, N.R. (1962) Skin resistance and galvanic skin response. *Arch. Gen. Psychiat.* 7 163-169.

Animal work has not yielded any clear information about the central control of electrodermal functioning either, with one interesting exception. It will be recalled (p.44) that spontaneous electrodermal responses are especially prevalent in slow-wave sleep (stages 3 and 4) in the human. Bloch and Bonvallet (1959)¹⁵ showed that in the un-anaesthetised cat, decortication produced large and regular electrodermal activity. They concluded that the cortex could exert an inhibitory effect on electrodermal activity. It would seem reasonable to suppose that the high spontaneous activity in humans in slow-wave sleep might result from reduction in such cortical inhibition.

Given that electrodermal responses are at least partially dependent on properties of an epidermal membrane, it is to be expected that measures of response may be affected by electrode and electrolyte properties. Edelberg et al (1960)¹⁶ and Edelberg and Burch¹⁵ (1962) have both described the effects of various electrolytes in strong concentration on observed amplitude of SRRs. Anion and cation effects were interpreted in terms of their effects on an active membrane, carrying a relatively low charge, when the ions are moved by the current used in measuring resistance. Edelberg and Burch concluded that a weak saline electrolyte was to be recommended, since this most closely approached the composition of natural sweat.

In skin resistance measurement the electrodes used in conjunction with the electrolyte may act as a battery. This battery effect will occur also in skin potential recording. In skin resistance measurement, /

¹⁵ Bloch, V. and Bonvallet, M. (1959) Contrôles cortico-reticulaire de l'activité électrodermale (réponse psychogalvanique) J. Physiol. (Paris) 51, 405-406.

¹⁶ Edelberg, R., Greiner, T. and Burch, N.R. (1960) Some membrane properties of the effector in the galvanic skin response. J. Appl. Physiol. 15, 691-696.

/measurement, however, these problems are multiplied. Passage of the recording current will "charge" the battery, and as long as current is passed the voltage produced (back e.m.f.) will vary (Lykken, 1959¹⁷). Such a system will have an internal resistance, which may change through the discharge of ions, and is thus a function of area of electrodes, current and time. These internal resistive effects and the back e.m.f. will clearly affect observed resistances.

In the measurement of skin potential level, the resistive effects are no longer important, but the battery effect or 'bias potential' remains a problem. The bias potential clearly should be as low as possible, and stable. This problem, especially that of fluctuations in the bias potential, has been discussed by O'Connell et al (1960)¹⁸. In the recording of skin potential responses, the stability of bias potentials in the absence of a "charging" current is the only issue of importance. For this purpose, inert electrodes are preferable, and the pure silver (not commercial silver, contaminated by copper) electrodes in use for electroencephalography are very suitable (Margerison et al. 1967¹⁹). Such electrodes should produce bias potentials which do not vary by more than about 1 microvolt per second.

¹⁷Lykken, D.T. (1959) Properties of electrodes used in electrodermal measurement. J. Comp. Physiol. Psychol. 52 629-634.

¹⁸O'Connell, B.N., Tursky, B. and Orne, M.T. (1960) Electrodes for the recording of skin potential. Arch. Gen. Psychiat. 3 252-258.

¹⁹Margerison, J.H., St. John-Loe, P. and Binnie, C.D. (1967) Electroencephalography. In Venables D.H. and Martin I. (Eds) A manual of psychophysiological methods. North-Holland, Amsterdam.

The discussion above clearly indicates that for recording an electrodermal component of the "orienting reaction", the measurement of skin potential responses presents the fewest problems. Electrode artifacts are reduced to a magnitude below the sensitivities of recording (SPRs being of 100 microvolts or more). There remain the problems of temperature and humidity. Air-conditioning should clearly reduce variation in these factors to a minimum. The results of Venables and Sayer¹⁴ enable us to calculate the likely effect of any variations in temperature. Using one active and one inactive electrode (on an abraded fore-arm site), skin potential variations of 0.1 millivolt within 5 seconds will only occur if skin temperature varies by more than 1 degree C in 10 sec. Such variation is unlikely in sleep except during body movements, when SPRs are apt to be artifactual in any case.

(b) Cardiac responses

Heart rate changes have long been recognised as one of the autonomic responses to external stimuli. For instance, Robinson and Gantt in 1947²⁰ described the cardiac and other components of the orienting reflex in dogs. Lacey has made an extensive study of heart rate changes in human subjects to a variety of stimuli (1950, 1956, 1967; Lacey et al. 1964). Davis et al. (1955) early described heart rate responses in human subjects as a component of the 'orienting reaction'. Graham and Clifton (1966) have reviewed a large amount of data in the ten years prior to their paper on heart rate changes as components of the orienting reaction; their paper has already been discussed earlier in this chapter.

²⁰ Robinson, J. and Gantt, W.H. (1947) The orienting reflex (questioning reaction): cardiac, respiratory, salivary and motor components. Bull. Johns Hopkins Hospital 80 231-253.

Lang and Hnatiow (1962) examined various measures of the heart rate response and suggested a measure which they regarded as most suitable for use in studies on habituation, given the problem of selecting from a mass of data a meaningful response measure. More recently, heart rate has been used in studies of the effect of various levels of "arousal" on responses to unconditional stimuli (McDonald et al. 1964), and the characteristics of heart rate responses in various stages of sleep was elucidated by Hord et al. (1966). Heart rate responses habituate normally in the alert, normal subject, in contrast to vasomotor responses, which may not (Furedy, 1968), and therefore heart rate responses provide a useful measure in studies of habituation in relation to state. If a cardiovascular measure is desired to complement an electrodermal measure in studies of autonomic responses, heart rate responses are therefore an obvious choice.

Heart rate is controlled in the first instance from the medulla. Sympathetic control of heart rate originates in the vasomotor centre of the medulla oblongata. 'Pressor' areas here receive innervation from cortex and hypothalamus, from the respiratory centre and from the chemoreceptors, principally. Stimulation of the pressor centre leads to an increase in heart rate and blood pressure, other factors being constant. The depressor area receives innervation principally from cortex, hypothalamus and the baroreceptors: stimulation of this area leads to an inhibition of sympathetic activity and a decrease in heart rate and blood pressure. The dorsal motor nucleus of the vagus initiates/

/initiates a tonic parasympathetic discharge which has an inhibitory effect on heart rate. This 'cardioinhibitory centre' is principally innervated by the baroreceptors, and by impulses from higher in the central nervous system (Uvnas, 1960²¹).

A variety of areas in the cerebral cortex have an effect on cardiac function. These include the tip of the frontal lobe, the anterior part of the temporal lobe, the orbital, cingulate, motor and premotor cortex (Delgado, 1960²²). Unfortunately, this anatomical information, especially in the absence of information on the function(s) of these areas, tells us little about these factors which affect heart rate in the intact organism, despite information as to the directions of such effects (for instance, stimulation of the motor cortex leads to an increase in heart rate in man). No systematic distribution has been found for the patterns of effects produced by cortical stimulation. The role of the active compensatory mechanisms of cardiac control complicates the picture further. Similarly the role of the hypothalamus is still unclear; while the hypothalamus appears to mediate certain cortical influences, Schaeffer (1960²³) concluded that the hypothalamus does not play a decisive role in cardiovascular homeostasis.

²¹Uvnas, B. (1960) Central cardiovascular control. In Field, J., Magoun, H.W. and Hall, V.E. Handbook of physiology. Section 1, Neurophysiology. Vol. 2. 1131-1162. American Physiological Society, Washington.

²²Delgado, J.M.R. (1960) Circulatory effects of cortical stimulation. Physiol. Rev. Suppl. 4. 146-171.

²³Schaeffer, H. (1960) Central control of cardiac function. Physiol. Rev. Suppl. 4. 213-231.

The effects of a number of physiological factors on heart rate is more clear. Internal temperature apparently has little or no influence on innervation to the heart from the medullary centres (Schaeffer, 1960). However, temperature acts directly on the sinoatrial node to increase heart rate: fluctuations in internal body temperature would therefore alter heart rate.

Motor activity is associated with an increase in heart rate. The increase in heart rate usually outlasts the movement, acceleration starting slightly prior to the actual movement; heart rate returns to baseline slowly after the movement. Such a response is illustrated in Fig. 3c.

Respiration and heart rate are also associated. Sinus arrhythmia is the obvious example of this. Sinus arrhythmia is produced in part by the stretch receptors of the lung, which on inflation of the lung cause acceleration of heart rate via the vasomotor centres. It is also produced by irradiation of impulses from the respiratory centre to the vasomotor centre (Brener, 1967²⁴). The heart rate response to external stimuli may therefore be modified by the presence of sinus arrhythmia. Since the heart rate response lasts about ten seconds, unless respiration rates are abnormally slow, sinus arrhythmia will be superimposed upon heart rate responses, and will thus increase the variance of measured heart rate responses. The problems which this presents in establishing whether habituation is taking place will be discussed in Chapter 9. In particular, decreases in breathing rate tend to increase the variance of heart rate, //

²⁴Brener, J. (1967) Heart rate. In Venables, P.H. and Martin, I. A manual of psychophysiological methods. North-Holland, Amsterdam, 103-131.

/rate, and vice versa, without changing mean heart rate (Engel and Chism, 1967²⁵).

Changes in blood oxygen and carbon dioxide also have an effect on heart rate. There is still doubt as to whether oxygen lack, or high carbon dioxide levels, are important in affecting heart rate and blood pressure, but it would appear that oxygen want leads to an increase in heart rate independent of the form of respiration, and high levels of carbon dioxide lead to a decrease in heart rate (Schaeffer, 1960²³). It follows that small variations in oxygen consumption should therefore have little effect on heart rate, as these two effects will act antagonistically. It would seem that if decreases in blood oxygen lead to an increase in heart rate, as Brener (1967²⁴) maintains, this effect must be a secondary result of increases in respiration, and not a direct effect. MacLeod and Scott (1964²⁶) have shown that in the cat, increases in heart rate following lowering of blood oxygen are certainly secondary effects, and that the primary response is bradycardia. As will be noticed from this discussion, however, the literature on this point is not fully consistent. There also appear to be marked inter-species differences. Thus in the cat, heart rate in REM sleep falls, whereas in man, the increased cerebral oxygen consumption in REM sleep is associated with an increase in heart rate and heart rate variability (Snyder et al. 1964; Brebbia and Altschuler, 1965²⁷).

²⁵Engel, B.T. and Chism, R.A. (1967) Effect of increases and decreases in breathing rate on heart rate and finger pulse volume. *Psychophysiology* 4 83-89.

²⁶MacLeod, R.D.M. and Scott, M.J. (1964) The heart rate response to carotid body chemoreceptor stimulation in the cat. *J. Physiol. (Camb.)* 175 193-202.

²⁷Brebbia, D.R. and Altschuler, K.Z. (1965) O₂ consumption rate and electroencephalographic stage of sleep. *Science* 150 1621-1623.

Blood pressure and heart rate are intimately related, via the various arterial baroreceptors in the carotid sinus, aortic arch and elsewhere. Increases in arterial pressure act via the vasomotor depressor areas and the dorsal motor nucleus of the vagus to decrease heart rate, and vice versa. Relatively little is known, however, about changes in blood pressure during sleep (as it is obviously not an easy variable to measure in sleep). About all that can be said is that blood pressure drops abruptly at the onset of sleep, and there is a fairly precise correlation between onset of sleep as judged by EEG criteria and the fall of blood pressure (see Snyder, 1971). It seems likely that variations in blood pressure during sleep are chiefly determined by variations in vascular resistance. Since these changes will be mediated by the vasomotor centres, it would seem unlikely that in sleep there are variations in blood pressure which are not components of reactions involving simultaneous changes in heart rate.

Baust and Bohnert (1969²⁸) have made a study concentrating on the regulation of heart rate during sleep. This study was done on unrestrained cats, not on man, but one can suppose that the mechanisms, if not the specific effects, are likely to be similar in man. Baust and Bohnert concluded that the fall in heart rate during slow-wave sleep is caused mainly by an increase in parasympathetic activity. The tonic fall in heart rate in the cat at the onset of REM sleep was induced primarily by a decrease in sympathetic activity. Baust and Bohnert further showed that this effect was not controlled by factors such as temperature or blood oxygen and carbon dioxide.

²⁸Baust, W. and Bohnert, B. (1969) The regulation of heart rate during sleep. *Exp. Brain Res.* 7 169-180.

In the cat bursts of eye movements in REM sleep are associated with an increase in heart rate commencing about a second before the EM burst, with a bradycardia following the burst in 70% of cases. Baust and Bohnert managed to show that these changes were primarily brought about by vagal influences, but changes in sympathetic activity also played a part.

In man, however, although a number of physiological variables have been shown to vary with the occurrence of eye movement bursts, including respiration and arterial oxygen, heart rate changes do not occur in conjunction with EM bursts (Aserinsky, 1965b²⁹; Spreng et al. 1968). Spreng et al. found that respiration rate and finger pulse responses were significantly altered during EM bursts, but electrodermal activity, heart rate and respiration amplitude were not. This enhances the suitability of heart rate over finger pulse responses as measures to be used in studies of responses to stimuli in REM sleep.

Possible artifacts affecting heart rate responses in sleep would therefore be phasic changes in body temperature, respiration, blood oxygen and carbon dioxide levels, and body movements. The discarding of data obtained during body movements is a simple process. Variability of the other functions is generally small in NREM sleep, but increased in REM sleep (Jouvet et al. 1960; Aserinsky, 1965; Snyder, 1971). There is no evidence that phasic fluctuations in body temperature/

²⁹Aserinsky, E. (1965)b Periodic respiratory pattern occurring in conjunction with eye movements during sleep. *Science* 150 763-766.

/temperature occur in REM sleep; fluctuations in blood oxygen and carbon dioxide, and respiration rates, associated with other phasic activity in REM sleep are known not to be associated with corresponding variations in heart rate. The one variable likely to artifact HRRs in sleep is therefore sinus arrhythmia. This problem will be treated in the discussion of results in Chapter 9.

CHAPTER FOUR

PREVIOUS STUDIES OF HABITUATION IN SLEEP

Can these responses to stimuli in sleep be habituated?

The number of such studies is not large. Moreover, almost all the studies published until about 1964 suffer from being descriptive and essentially qualitative, often relying heavily on a few individual examples. Moreover, there have been only two studies which have attempted to look systematically at responsiveness in each sleep stage or state - at least differentiating between REM and NREM sleep. All of the other studies have looked exclusively at NREM sleep, and presumably most of these looked at stage 2 sleep shortly after sleep onset. In several studies in the 1950s it was regarded as quite acceptable to induce sleep with barbiturates, ignoring any effect this might have on responsiveness to stimuli.

Pampiglione in 1952 in a brief note was the first to report that K-complexes habituated in sleep, although he reported that this only occurred if the interval between stimuli was less than 10 seconds. In 1954 Schwab et al reported that habituation of EEG K-complexes to repeated stimuli in sleep was possible, although they reported that habituation only sometimes occurred. On occasions with short interstimulus intervals (ISIs) K-complexes 'summed' to produce an arousal reaction after three or four stimuli. However, when repeated stimulation allowed uninterrupted sleep, Schwab agreed with Pampiglione that habituation tended to occur when ISIs were less than 10 sec, whereas habituation did not on the whole occur when ISIs/

/ISIs were 10 sec or longer. Pampiglione and Ackner in 1958 confirmed that habituation (or 'adaptation' as they called it) occurred in natural sleep as well as in the barbiturate induced sleep of their earlier studies. They found that both EEG K-complexes and vasomotor responses habituated, although they noted that the two responses behaved independently, or were 'dissociated'.

In 1956 Roth et al claimed that adaptation of K-complexes did not occur unless the interval between stimuli was less than 2 seconds - in other words, that habituation of K-complexes was very difficult to achieve at all. Roth's study was however criticised by Pampiglione and Ackner (1958) on the ground that Roth et al. presented their initial stimuli shortly after the administration of barbiturates, when it is difficult to elicit K-complexes. Thus Roth et al were unable to detect habituation because they had reduced initial responsiveness. Also, Roth et al used click stimuli (in contrast to the more commonly used tone stimuli). Clicks always include sudden high frequency transients, potent in producing startle reactions in awake subjects. It seems at least likely that the K-complexes evoked by Roth et al's clicks were more 'startle' than 'orienting' responses (if such a distinction is possible in sleep), and as such may have been less readily habituated.

Oswald, Taylor and Triesman (1960) reported that habituation of K-complexes did occur to the presentation of names spoken over a tape recorder, but they merely stated that habituation "has been observed, being much more marked in some subjects than others";

/others; they provided no numerical indication of the extent or speed of habituation. Oswald et al pointed out that since K-complexes discriminate between meaningful and non-meaningful words, the failure of response must occur after cortical analysis, not merely as a result of peripheral sensory inhibition or fatigue.

Beh and Barratt (1965), as part of their study on the conditioning of K-complexes, also found that K-complexes habituated to repetitive stimulation. They found that the K-complexes to the conditional stimulus (a mild shock) extinguished if the mild shock was not associated with the conditioned stimulus.

Sharpless and Jasper in 1956 made a now classic study on the habituation of the arousal reaction in the sleeping cat. That the 'tonic' arousal reaction (lasting several minutes) habituated is not of concern here, for such an awakening is quite different from the EEG and autonomic responses considered here. But their 'phasic' reaction, more like the sort of EEG responses considered by other authors, was much more resistant to habituation, taking some 30 trials to extinguish completely. Sharpless and Jasper found that habituation became faster as the ISI shortened, although this variable was not properly controlled in their study. They also reported that habituation was faster in 'deeper' sleep, but did not give any numerical substantiation of this. Sharpless and Jasper's other finding was what they called 'experimental habituation': habituation in the second and third days of the experiment occurred more readily than on the first.

In 1961, Sokolov and Paramonova reported habituation of vasomotor, respiratory, GSR and EEG components of the OR in sleep. Their study suffers from being essentially qualitative. However, they managed to conclude that the GSR habituated (or was 'extinguished') most readily, within roughly 5 to 10 trials. The response most resistant to extinction was the EEG reaction, "which merely showed a gradual reduction in strength and duration, and persisted up to the 23rd application" of the stimulus. Unfortunately Sokolov and Paramonova did not specify what they meant by EEG reaction: their term appears to have included both K-complexes and also more prolonged arousal reactions, with the appearance of alpha for up to 10 seconds. Thus their results for EEG responses are not comparable with those of other authors except possibly Sharpless and Jasper. One general finding Sokolov and Paramonova made was that they could not reliably produce dishabituation (or disinhibition) by the use of a novel acoustic stimulus unless it was of greater intensity than the habituating stimulus. Although not specified, it would appear that Sokolov and Paramonova confined their study to stage 2 NREM sleep, as they were especially interested in the development of sleep.

Oswald (1962, p.30 ff) reported that if (NREM) sleep deepened as stimuli were continued, the GSRs elicited became dissociated from the stimuli. Although this report has been taken by Johnson and Lubin (1967) to imply that there was no habituation in medium or 'deep' sleep, this inference is incorrect: as deep sleep supervened, stimuli no longer elicited GSRs. The GSRs observed then occurred randomly and spontaneously.

In a much more recent study also confined to NREM sleep, Tizard (1968) reported no habituation of either SPR or EEG K-complexes in 8-10 year old children. In this study twenty tones were presented at random ISIs of between 20 and 40 seconds. In an earlier study, however, Tizard (1966a, 1966b) had found SPR habituation in adults during a study of the development of sleep. In that study stimuli were presented at regular intervals of 20 sec, instead of at irregular intervals. However, the interpretation of these results is a little unclear, since results for both waking and sleeping were combined, and habituation was found in the total sample. In these 1966 studies, she also reported on EEG responses, again combined for both sleep and waking, and found no habituation.

Tizard's failure to find habituation of the SPR when using irregular ISIs of between 20 and 45 sec. is striking, for Davis (1970) has made an estimate of what he terms the 'refractory period' of an unconditioned response as being about 60 seconds. This 'refractory period' represents the maximum time interval between successive stimuli over which a short term habituation process could operate. Thus use of ISIs approaching this period would be expected very much to reduce the possibility of detecting habituation in sleep even if habituation were possible with shorter ISIs.

By contrast with the other authors cited so far who looked only at NREM sleep, McDonald and Carpenter (1966) studied habituation in all stages of sleep systematically, REM as well as NREM sleep stages. This study is an extension to all stages of sleep of the study of/



/of McDonald et al in 1964 on habituation in alert and drowsy subjects. In this study, they divided their subjects into two groups on EEG criteria: their 'drowsy' group included those who showed evidence of sleep on at least one (of ten) trials. Their subjects' state fluctuated readily between wakefulness and stage 1 and even stage 2 sleep. Essentially, they found that cardiovascular response habituation was blocked in their 'drowsy' subjects, although electrodermal response habituation was unaffected by state.

In their 1966 study, McDonald and Carpenter, using ISIs between 10 and 20 seconds, found "consistent, significant habituation" of both heart rate responses and finger plethysmographic responses (FPRs) in all EEG stages of sleep. Skin potential responses showed significant habituation in only about half of their tests and they therefore were "inclined to minimise the habituation of this measure". Skin resistance responses (GSRs) did not occur in sleep at all in their study. It is worth remarking in this context that although McDonald et al (1964) found no habituation of the HRR in drowsy subjects, as mentioned earlier, McDonald and Carpenter did find habituation of the HRR in sleep stages 2, 3, 4 and REM, while confirming the earlier results on drowsy subjects (stage 1). This study unfortunately still unpublished, is particularly important as it is one of the only two studies yet to have looked at habituation in all stages of sleep individually.

The other such study is that of Johnson and Lubin (1967).

Using ISIs between 30 and 45 seconds, Johnson and Lubin reported finding no habituation of any of their measures, SPR, CSR, HRR, FPR, respiratory responses and EEG K-complexes. They said that "our data indicate that sleep blocks habituation in the case of autonomic variables, and evoked K-complexes ... habituation and sleep were incompatible". It is my contention, however, that what Johnson and Lubin were actually studying in sleep was not habituation at all, but a time of night effect. They quote Williams et al (1964) in support of their own findings, as showing no habituation of either the FPR or EEG K-complexes during a whole night of sleep. Williams et al themselves say, however, that they were studying the effects of sleep stage, sleep deprivation and time of night on responsiveness, and they do not claim to have studied habituation. Johnson and Lubin plotted per cent responses to stimuli for SPR, CSR, FPR and HRR against hour of the night, averaging the response rates over hourly periods. Stimuli were presented continually all night, starting before the subjects went to bed. Sharpless and Jasper (1956) suggested that in sleep, arousals are crucial in producing dishabituation. Because Johnson and Lubin averaged responses over hourly periods in the night, dishabituation following arousals will necessarily have obscured any habituation that might have been present. This should be clear if one considers what meaning has the mean stage 2 response for an hour of sleep comprising a sequence of stages such as 23423231212342! For comparison with these hour-by-hour curves for sleeping subjects, Johnson and Lubin presented trial-by-trial curves of responses in day-awake and in pre-sleep waking subjects. These curves show/

/show clear habituation of all the variables over the course of 10 trials in the pre-sleep conditions; but they cannot validly be compared with mean responses plotted hour by hour in sleep. All mean hourly response rates can show is a time of night effect.

Johnson and Lubin also quote Hord et al (1966) as showing that the heart rate response did not habituate in sleep; however, Hord et al merely state (i.e. assume) that heart rate response are non-habituating in sleep: they offer no evidence.

However, Johnson and Lubin did make a brief mention of another study in which subjects were presented with ten tones in stage 2 sleep without any prior stimulation. HRRs, GSRs and FPRs were recorded, and no significant habituation was found for any of the three variables, although no numerical data is presented from this experiment.

One other paper has examined habituation in both NREM and REM sleep; these authors (Martinius and Papousek, 1970) reported finding habituation in NREM sleep but none in REM sleep. Specifically, they found habituation of eye blinks to either visual stimuli or air puffs in the NREM sleep of young babies, but they also reported the occurrence of a response which did not habituate in REM sleep. Unfortunately Martinus and Papousek's paper gives little specific information by which one could evaluate their findings. Their criterion for habituation was 5 consecutive failures of responses. They quote a mean number of twenty trials to habituation in NREM sleep (the range was from 3 to 46 trials) but they give absolutely no data for REM sleep. They merely state that "the infants showed the blink/

/blink reflex with varying intensity ... habituation was reached incompletely or temporarily only". So their findings are impossible to compare with those here; they offer only the sentence quoted above and two illustrative figures to describe events in REM sleep. Furthermore, sleep states were assessed visually: no EEG criterion of sleep state was used, which renders their results of somewhat dubious value.

Purpose of the present experiments:

The purpose of these experiments was to try and clarify whether habituation was possible in human sleep, given stimulus conditions which would be conducive to habituation in waking. Habituation in sleep was taken to mean the waning of responses to repetitive stimuli within ongoing sleep, rather than habituation of "arousal" reactions which include the awakening of the sleeper. When this study was begun, I was unaware of the as yet unpublished work of McDonald and Carpenter referred to already. It therefore seemed important to remedy the defects of the one study to that date which had attempted to look at habituation in sleep systematically.

CHAPTER FIVE

DESIGN AND METHOD

1. Design of the study:

The overall intention was to use a design in which each series of trials presented to each subject under each condition could be analysed separately. The object was to determine whether habituation had occurred in each series of trials, and to make tests for statistical significance separately for each series. Subjects would be used as their own controls: they were to be put through all conditions. The objective of assessing habituation separately in each series of trials was in practice only possible for the autonomic responses, for which a numerical estimate of magnitude could be made for each subject on each trial. EEG responses had to be treated in terms of their frequency, and had to be combined for all subjects.

Two autonomic responses were chosen for study: the skin potential (Tarchanoff) response, and the heart rate response. The SPR and HRR were chosen to provide one electrodermal and one cardiovascular response measure. In addition, EEG responses were studied in NREM sleep, and in REM sleep. The EEG response to a stimulus in NREM sleep, the K-complex, has been described already; it is clearly visible in stage 2 sleep. In stage 4 sleep it is obscured by the prevailing high voltage slow wave activity, so there is no measure of EEG response available without averaging techniques. It was desired to choose a response in REM sleep for comparison, and the transitory appearance of alpha rhythm in response to a stimulus provides such a response (Fig. 2). The comparable response in waking, namely, alpha blocking, could not be used, since subjects were asked to/

/to keep their eyes open during daytime controls, and consequently alpha activity was not plentiful in the EEG to start with.

Autonomic responses were studied in daytime "controls" (which were intended to be waking controls, but which ended as daytime "controls" in states of drowsiness), in NREM sleep stages 2 and 4, and in REM sleep.

Sleep stage or state was one of the two chief variables of the experiment; the other was interstimulus interval (ISI), chosen because it was thought that ISI might be critical in determining whether one could detect habituation in sleep. Three ISI ranges were employed: 10, 20 and 30 seconds. A total of six ISI conditions were provided by using three conditions with those regular intervals, and three conditions with irregular intervals around those mean values. For these conditions the intervals 8, 10 or 12 seconds, 16, 20 or 24 seconds, and 24, 30 or 36 seconds (for the three irregular conditions respectively) were presented in random order.

Because the object was to analyse each series of responses for each subject separately, and to use subjects as their own controls, only three subjects were employed. They each underwent all 24 conditions: 4 sleep stages or states under each of the six ISI conditions. Only one condition was presented in any one night, and all experimental conditions were separated by weekly intervals. Order of presentation of conditions (Table 2) was balanced for order effects (excepting cyclical order effects) except in two respects. First, daytime controls always preceded the sleep conditions for any given ISI condition. Second, all the regular stimulus interval conditions preceded all the irregular conditions. The experiment/

/experiment was in a sense conducted as two experiments whose results were then pooled.

2. Quantification of the data:

Sleep stages were scored in 20 second epochs according to the standard criteria of Rechtschaffen and Kales (1968), except that movement time was not scored.

Skin potential responses commencing with a latency from stimulus onset of between 1.0 and 3.5 seconds were scored in mV. The difference between the peak negative and subsequent peak positive potentials was used to define the magnitude. Geer (1966) has shown that among latency, duration and amplitude measures of the GSR, amplitude was the most sensitive to the effects of stimulus repetition.

The measure of heart rate response used was derived from Lang and Hnatow (1962). It measures the difference between the peak of the acceleration following the stimulus, and the trough of the subsequent deceleration. Since there may be several distinct peaks and troughs, the measure was actually defined as the maximum difference between a peak and a subsequent trough in the ten beats following stimulus onset.

EEG responses: not being readily quantifiable, were scored 'blind' by an experienced independent judge. The judge was presented with sections of EEG (each lasting 40 sec), randomly selected from the whole series of stimuli, in such a manner that he could not tell whether a stimulus came at the start or end of a series. The judge scored the response into three categories. The first was a fully developed response (K-complex in stage 2 or alpha-burst in/

/in REM sleep); the second category was some EEG response but not a fully developed response; the third category was no response.

The criteria for K-complexes were those employed by Oswald et al (1960), a fully developed K-complex consisting of an obvious large slow wave with associated 12 HZ activity, with a latency of not more than 2 seconds from stimulus onset. A slow wave without associated 12 HZ activity was scored as a 'response' but not as a K-complex.

For REM sleep a visible burst of alpha activity (9-11 HZ) with a latency of no more than 2 seconds was scored as an 'alpha response'. A small spike wave or a 'flattening' of the EEG (reduction in voltage) was scored as a 'response' but not as an 'alpha response'.

Although these categories might be regarded as comprising an ordinal scale (the scale for K-complexes was designed as an ordinal scale), both scales were treated as purely nominal, representing initially three categories of response A, B or none. But since EEG responses other than fully developed K-complexes or alpha responses were seen on initial inspection of the data to be uniformly distributed throughout all trials, it was decided at the outset of the data analysis to compress the EEG response categories from three into two: the presence of a fully developed K-complex or alpha response, or their absence. Partly developed EEG responses were ignored. An advantage of this is that the handling of dichotomous/

/dichotomous data is easier.

Artifacts: Individual autonomic responses occurring simultaneously with a body movement were discarded as artifact, since movements often produce large SPRs and HRRs whether or not a stimulus has been presented. One such large HRR is illustrated among the records in Figure 3(a) where a stimulus led to a movement and arousal.

Another example occurs at the end of the record in Fig. 3(c).

A body movement was defined as one in which phasic muscle activity appeared in the EMG channel, and in one of the EOG, EEG or SPR channels. Also some HRRs had to be discarded as artifact owing to ratemeter malfunctioning. The number of such responses is given at the foot of Table 3.

Spontaneous variability: A 5 minute period before each presentation of a series of stimuli was used to obtain an indication of 'spontaneous response' rates, or a control for spontaneous variability of the measures employed. During this 5 minute period, ten pseudostimuli were marked on the record. These are marks on the record where no stimuli actually occurred. 'Responses' to these 'stimuli' are then scored just like all other responses; these 'pseudoresponses' thus provided a measure of spontaneous autonomic and EEG activity. Unlike Johnson and Lubin (1967) who subtracted the pseudoresponses from the actual responses, in this study the pseudoresponses provided a 'control' rate of activity against which habituation could be compared.

The points at which pseudostimuli were marked were chosen to 'mimick' the actual ISI condition used in the stimulation period.

For instance, when the 30 second irregular ISI condition was to be used, the ten pseudostimuli were marked at randomly chosen intervals of 24, 30 or 36 seconds, commencing at a point 5 minutes before the actual start of stimulation.

3. The data analysis:

The method of analysis chosen for the autonomic data was that of individual regressions for each response series¹. The regressions fitted were linear regressions in two segments (Fig. 4). Each series of responses was fitted to two linear segments by means of a computer program. Then it was established for each response series whether the two segment regression fitted the data significantly better than the null hypothesis. The null hypothesis was that the response magnitude did not change with trial number, i.e. that the mean response was the best estimate of the response magnitude, independent of trial number.

Two parameters were extracted from the regressions as measures of 'rate' of habituation, namely, the slope of the first segment of the regression and the turning point (how many trials it takes for habituation to be 'complete'). These measures were each subject to a repeated measures analysis of variance. Thus it was hoped to determine whether there were any significant effects of either ISI or state (sleep stage) on habituation. State and ISI were the two within-subject factors. Orthogonal comparisons were used following these analyses where the F test was significant (Edwards, 1968).

¹The regression is described in greater detail on page 72.

The distribution of turning points was markedly skewed, as can be seen from a glance at Tables 10 and 11. Although Scheffe (1959) points out that a lack of normality in distributions does not seriously affect the validity of analyses of variance, a logarithmic transformation was applied to the turning points before the analysis. This transformation also helped to reduce the differences in variance between the various conditions. Although there remained a significant variance ratio between different states (between 2.5 and 3.5), Scheffe (1959, p353) has pointed out that when cell sizes are equal the effects of unequal error variances are negligible.

In addition to the linear regressions described above, a purely graphical approach was adopted. Mean responses were averaged over all subjects and ISIs for each state as a function of trial number, to illustrate the form of habituation in each state. A comparison of the means of the fitted slopes and turning points with these graphs should also provide a 'face validity' for the two segment regression used.

EEG responses could not be subject to the regressions of magnitude on trial number. Instead the frequency of EEG responses was calculated for all subjects in blocks of trials; habituation and the effect of ISI were then examined graphically.

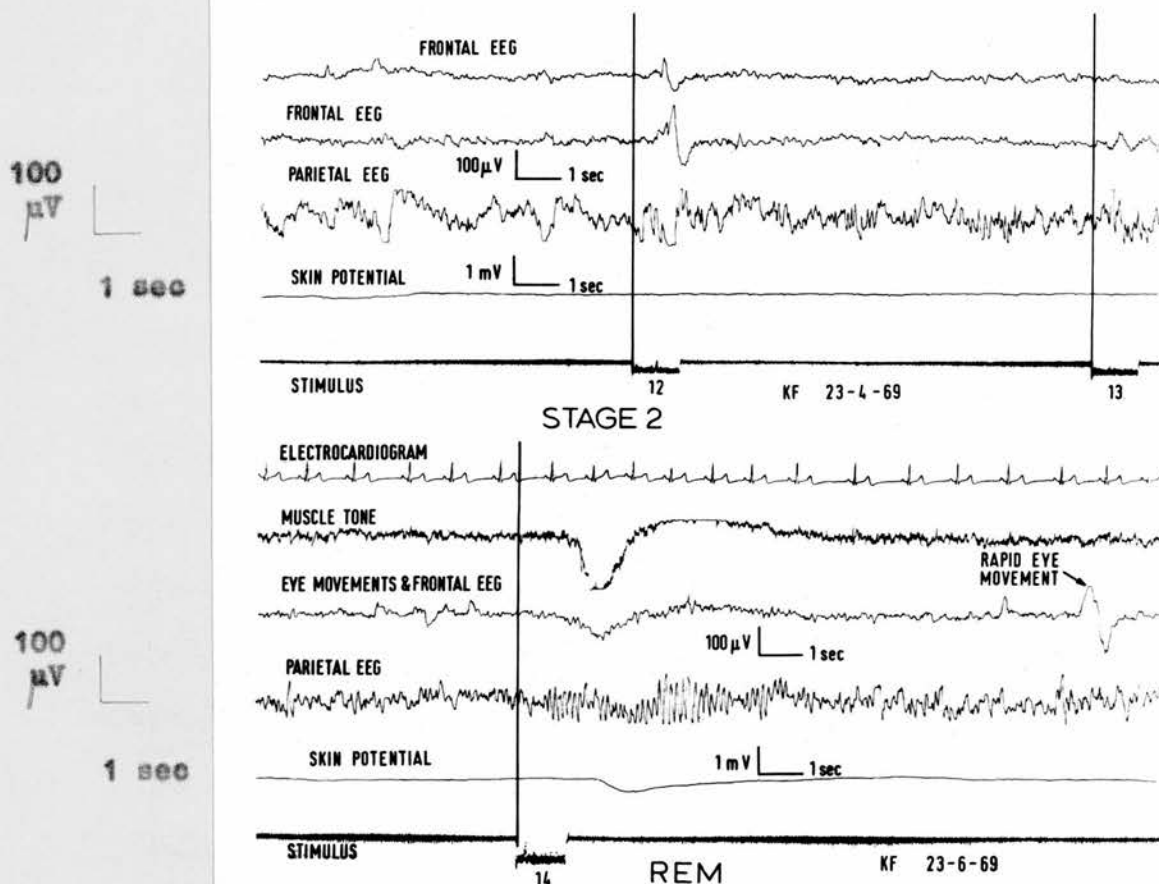
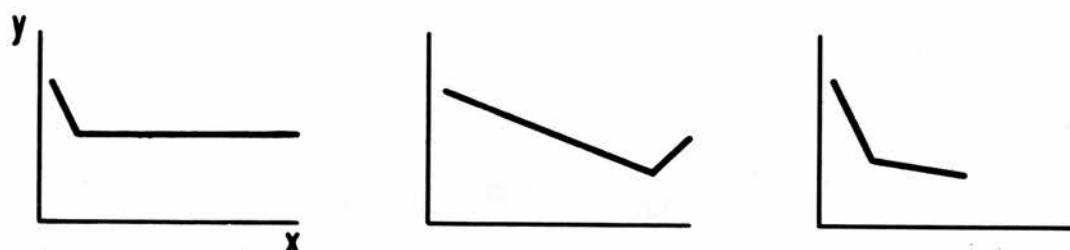


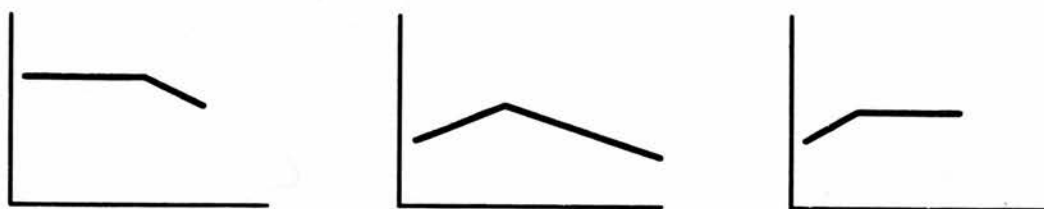
FIGURE 2. AN EVOKED K-COMPLEX IN STAGE 2 (UPPER) AND AN EVOKED 'ALPHA RESPONSE' IN REM SLEEP (LOWER).

Calibrations in the figure refer to skin potential, and to frontal EEG and eye movement channels. Calibration for parietal EEG is indicated alongside.

TWO SEGMENT LINEAR REGRESSION EXAMPLES



PREDICTED BY HABITUATION HYPOTHESIS
SLOPE OF FIRST SEGMENT NEGATIVE



CONTRARY TO HABITUATION HYPOTHESIS
SLOPE OF FIRST SEGMENT ZERO OR POSITIVE

FIGURE 4. EXAMPLES OF TWO-SEGMENT LINEAR REGRESSIONS.

The upper set are three possible examples all of which involve initial response decrement. The lower set are examples where there is no evidence of response decrement.

TABLE 2.

ORDER OF CONDITIONS: REGULAR ISIs

Subject 1	ISI	State	ISI	10	20	30
	10	1 Daytime	1	1	5	9
	20	2 REM	2	2	6	10
	30	3 NREM 2	3	3	7	11
		NREM 4	4	4	8	12
Subject 2	10	2 Daytime	1	5	9	1
	20	3 REM	3	7	11	3
	30	1 NREM 2	4	8	12	4
		NREM 4	2	6	10	2
Subject 3	10	3 Daytime	1	9	1	5
	20	1 REM	4	12	4	8
	30	2 NREM 2	2	10	2	6
		NREM 4	3	11	3	7

Irregular ISI conditions were presented subsequently,
in the same order as the regular ISI conditions above.

TABLE 3.

A BREAKDOWN OF STIMULUS PRESENTATIONS

	DAYTIME CONTROLS	REM	STAGE 2	STAGE 4	TOTAL
Series analysed	18	18	18	18	72
Series of less than 20 trials discarded	2	22	11	15	50
Total series presented	20	40	29	33	122
Total trials in series analysed	469	489	632	504	2094
Total trials in series discarded	18	163	81	116	378
Total trials presented	487	652	713	620	2472
Mean length series analysed (trials)	26.1	27.2	35.1	18.0	29.1
Mean length of discarded series (trials)	9.0	7.4	7.4	7.7	7.6
Longest series analysed (trials)	49	50	50	50	50
Short series (less than 20 trials) analysed	-	10, 13, 19	14	10	-

Not included in this table are 5 daytime controls abandoned because the subject fell asleep. These sessions included 85 trials. In the series analysed, 139 HRRs and 50 SPRs (6.6% and 2.4%) were discarded as artifact owing to either gross body movement or (in the case of HRRs) machine artifact.

TABLE 4.

PRESTIMULUS HEART RATE:Summary of analysis of variance.

<u>Daytime Controls</u>					
SOURCE	SS	DF	MS	F	p
Subjects	2067.58	2	1033.79	100.81	.001
Trials	21.26	3	7.08	0.69	ns
Subjects x trials	61.52	6	10.25		
ISIs	1689.79	5	337.95	1.18	ns
Subjects x ISIs	2846.25	10	284.62		
ISIs x trials	232.15	15	15.47	1.29	ns
Subjects x trials x ISIs	357.30	30	11.91		
Total	7275.87	71			
<u>REM sleep</u>					
Subjects	2348.86	2	1174.43	111.99	.001
Trials	74.33	3	24.77	2.36	ns
Subjects x trials	62.91	6	10.48		
ISIs	377.11	5	75.42	0.42	ns
Subjects x ISIs	1775.97	10	177.59		
ISI x trials	293.00	15	19.53	1.04	ns
Subjects x ISIs x trials	560.25	30	18.67		
Total	5492.44	71			

TABLE 5.

PRESTIMULUS HEART RATE:Summary of analysis of variance.NREM Stage 2 sleep

SOURCE	SS	DF	MS	F	p
Subjects	5103.58	2	1551.79	112.66	.001
Trials	95.15	3	31.71	2.30	ns
Subjects x trials	82.63	6	13.77		
ISIs	363.95	5	72.79	0.36	ns
Subjects x ISIs	2016.08	10	201.60		
Trials x ISIs	180.43	15	12.02	1.52	ns
Subjects x trials x ISIs	237.02	30	7.90		
Total	6078.87	71			

NREM Stage 4 sleep

Subjects	4841.08	2	2420.54	267.72	.001
Trials	19.66	3	6.55	0.72	ns
Subjects x trials	54.25	6	9.04		
ISIs	377.33	5	75.46	0.55	ns
Subjects x ISIs	1369.08	10	136.90		
Trials x ISIs	89.66	15	5.97	1.04	ns
Subjects x trials x ISIs	170.91	30	5.69		
Total	6922.00	71			

CHAPTER SIX

A DISCUSSION OF PROBLEMS IN THE METHODOLOGY

1. Reasons behind the design

(a) The factors examined:

Sleep stages: The states chosen to study were stages 2, 4 and REM, with daytime controls. Stage 1 (drowsiness) was not initially chosen because of the difficulty of presenting a series of stimuli over an extended period without the subject oscillating either between drowsiness and wakefulness or slipping into stage 2 sleep or deeper. Also, habituation in alert and drowsy subjects had already been described in a very clear paper by McDonald et al (1964). In practice, however, it turned out that the daytime (supposedly alert) controls were drifting unsystematically between waking and drowsiness.

Stage 3 was omitted because it has already been shown that responsiveness does not differ significantly between stages 3 and 4 (Hord et al 1966; Johnson and Lubin, 1967); stage 3^{is} generally regarded, along with stage 4, as part of 'slow wave sleep'. Stage 3 is quite arbitrarily distinguished from stage 4 in terms of the percentage of slow wave EEG activity present; it was felt that the effect of increasing 'depth' of NREM sleep could be demonstrated sufficiently well by differences between stages 2 and 4.

Interstimulus intervals: Besides sleep stage or state, it was felt important to study the effect of another variable upon habituation: the interstimulus interval (ISI). This was considered important because the two studies which found no evidence of autonomic response habituation in sleep (Johnson and Lubin, 1967; Tizard, 1968) both/

/both used long irregular ISIs. Since habituation is slowed and ultimately abolished by increasing the interval between stimuli, it would be expected that use of long irregular ISIs would mitigate against finding habituation in sleep - especially if habituation were slower and therefore less detectable in sleep than in waking.

Two effects of ISI were thought relevant: first, the interval itself, and second whether this interval was regular or irregular. The range of ISIs practicable was limited on one side by the duration of the responses. Heart rate responses typically last for at least 10 sec, so in order to include conditions optimal for observing habituation, and at the risk of presenting stimuli before the heart rate could return to prestimulus levels, a lower limit for the ISI used was set at 10 sec. An upper limit of 30 sec was chosen to provide an ISI comparable with that used by Johnson and Lubin, but short enough to provide a reasonable number of stimuli in a given time. For instance, a typical REM period may last 15 to 30 minutes; however, REM periods are frequently broken up by NREM sleep for periods of several minutes at a time.

(b) Balancing of the design.

The order of presentation of conditions was partially balanced between subjects. Balancing was not achieved in three respects. First, the regular ISI conditions were all presented before any of the irregular ISI conditions. Thus the experiments were conducted in two parts, first with regular ISIs and then with irregular/

/irregular intervals. This was done so that some results could be assessed before the end of the series of experiments. The predicted difference between regular and irregular intervals was that habituation would be more difficult with irregular than with regular intervals. Any order effect in the experimental design would, however, have produced a tendency in the reverse direction: 'experimental habituation' (Sharpless and Jasper, 1956) would have made habituation easier later in the experiments.

The second feature lacking proper balance was the day-time controls, which for any given ISI preceded the sleep conditions (see Table 2). Here, too, however, any 'experimental habituation' would have led to faster habituation in the sleep conditions, whereas the predicted result was that habituation would if anything be slower in sleep than waking, if it occurred at all.

The third uncontrolled factor was cyclical order effects, both for ISI and for sleep stage. However there is no reason to expect cyclical carry-over effects, and moreover as a precaution against any such effects, all experimental sessions for each subject were separated by weekly intervals.

- (c) Subjects: Three subjects (two men and one woman aged between 18 and 25) were each put through all possible 24 conditions resulting from the use of 4 states and 6 ISI conditions. They were selected from volunteers who had heard that the laboratory needed subjects for sleep experiments. Selection was made of the first three subjects who were prepared to come regularly once a week over a period of/

/of approximately six months, to abstain from tea, coffee or alcohol on the days of experiments, and who were judged reliable enough (on informal interview) to achieve these conditions over a period of six months. Subjects were paid one pound for each attendance in the laboratory. Use of such a small number of subjects is not conventional in psychological research. The object however was to fulfil the advice given by Scholander (1960): "the great inter-individual variability of such (autonomic) responses makes it preferable to use an experimental design which involves only intra-individual comparisons". The use of such a small number of subjects is not on the other hand infrequent in sleep research (e.g. Hord and Ackerland, 1971; Akindele et al 1970), involving not only whole night recordings, but also the necessity of giving each subject nights of sleep from which data is not collected - so that he can adapt to sleeping in the laboratory situation.

Volunteers may differ from the general population. There is, however, no reason to assume that patterns of habituation might differ in volunteers and non-volunteers. (It should be borne in mind anyway that this experiment was a comparison between habituation in sleep with daytime controls). Although personality does appear to affect habituation, studies to date have not been consistent in their results. Eysenck's theory (1957, 1967) predicts that since habituation is related to the build up of reactive inhibition, extroverts should show faster habituation than introverts. Moreover neuroticism should be unrelated to habituation patterns.

However, what evidence there is, is contradictory (Mangan and O'Gorman, 1969). Martin (1960) for example found no correlation between extraversion and habituation rates. Further, habituation rates do seem to be related to anxiety though in which sense is not clear, as anxiety has been found to both increase (Sadler et al 1971) and slow down habituation rates (Lader, 1964; Lader and Wing, 1966; Coles et al 1971). Rosenthal (1971) reported no difference in habituation rates between his group of anxious subjects and his normal controls. However, anxiety appears to have been only a secondary symptom in many of his subjects.

Three studies of the personality characteristics of sleep volunteers have all shown no systematic personality biases likely to affect habituation. Tune (1968) found no differences in neuroticism scores between volunteers and non-volunteers. Lewis (1969) found that the only respect in which volunteers differed from population norms on Cattell's 16 PF inventory was on the intelligence factor. Lastly, twenty volunteers for other experiments described later in this thesis differed from the general population in neither extroversion nor neuroticism, as assessed by the MPI.

2. Data scoring and analysis.

(a) Heart rate scoring:

Heart rate responses(HRR) scoring was based on the method of Lang and Hnatow (1962). They tried two HRR measures based on the difference between the 'peak' rate following acceleration and the 'trough' rate after the subsequent deceleration. They first took the difference between the fastest beat in the five beats after/

/after stimulus onset and the slowest beat following it before the heart rate accelerated again. Their second measure used the fastest beat in the five following the stimulus and the slowest beat in the twenty beats following stimulus onset. They found that the latter measure showed the effects of stimulus repetition most consistently (in waking subjects). Hord et al (1966) have shown that in sleep the trough of the HRR occurs within about 10 seconds after stimulus onset. Since in this study it was intended to use ISIs as short as 10 seconds, a modification of Lang and Hnatow's HRR measure was used.

The HRR measure was defined as the maximum difference between a 'peak' rate and a subsequent 'trough' rate in the ten beats following stimulus onset. Lang and Hnatow's restriction that the 'peak' rate be taken from the first five post-stimulus beats was dropped as unnecessary. The more substantial departure from Lang and Hnatow's measure is the restriction that the 'trough' within the first ten beats only be scored. That this definition does pick up the trough of the HRR can be seen from Figure 5 in the results.

But with the short ISI conditions there is the likelihood that the heart rate will not have returned to prestimulus levels by the onset of the next stimulus. Thus responses to second and subsequent stimuli will be contaminated by the responses to previous stimuli. What effect this could have on HRRs is difficult to predict, since it depends on the recovery time of the HRR. Hord and Ackerland (1971) have published a paper since these experiments were actually completed in which they examined precisely this issue.

They reported that with an interval of 9-10 sec between stimuli, 'peak' responses to the second of the two stimuli were enhanced, 'trough' responses apparently unaffected. The HRR measure used here would thus be magnified on the second (and presumably subsequent) trials relative to its 'true' value. In an attempt to assess the effect of this complication in demonstrating habituation with short ISIs, a repeated measures analysis of variance on the rate of the immediately prestimulus beat was performed for each state (within-subject factors were ISI and trials). Prestimulus heart rate at trials 1, 6, 11 and 16 were used, for each ISI. The rationale for this was that if incomplete recovery of heart rate from previous stimuli was to affect responses significantly, then pre-stimulus heart rate would be significantly lower on later trials than on trial one. It was predicted that if recovery of the heart rate from initial stimuli was having a significant effect on HRRs to subsequent stimuli with the two short ISI conditions, then the effects of ISI and/or the interaction of trials with ISI on the prestimulus heart rate should be significant. In fact (Tables 4 and 5) there were no significant effects in any of the four states of ISI, trials, or their interaction. Hence it seems legitimate to accept the HRR results for the 10 second ISIs at their face value.

(b) Method of analysis chosen to demonstrate habituation of autonomic responses:

The chief criticism here levelled at the study of habituation by Johnson and Lubin (1967) concerned the technique of those authors in averaging responses over many trials. Habituation, while it/

/it may include long-term processes operating progressively over many experimental sessions, is normally considered as a short term process, as when a response may disappear in awake subjects in a few trials. As it was thought that habituated responses might in some subjects spontaneously return or dishabituate as they do in wakefulness (Sokolov, 1963a, p.119), it was felt necessary at the outset to employ a design in which each series of responses in each subject could be analysed separately.

It was predicted that habituation might take more trials in sleep than in wakefulness, as Sokolov and Paramonova (1961) reported; specifically, since Tizard (1968) had found no habituation of the SPR in twenty trials, it was thought possible that habituation might take more than twenty trials in at least some sleep stages, but very much less than twenty in the daytime (supposedly waking) controls. If Sokolov's (1960) model of OR habituation was essentially correct, and stage 4 slow wave sleep was indeed associated with reduced cortical inhibition, then habituation in stage 4 could be expected to be very slow indeed if it occurred. Fifty trials were felt necessary to be sure of detecting any such slowly occurring habituation.

The procedure for determining whether or not habituation was occurring had therefore to be capable of discriminating rapid habituation over as few as five trials, or alternatively very slow habituation taking up to fifty trials. It was hypothesised that the response measures used would decrease after a variable number of trials to some steady value representing purely spontaneous/

/spontaneous variability in the response measure. A two-segment linear regression was chosen to analyse each separate series of responses (see Figure 4).

Such a regression can be algebraically expressed as $y = ax + b$ for the range where x is less than z , and $y = cx + d$ for the range where x is greater than z . ' z ' is the 'turning point' of the regression, where the two linear segments meet. These regressions were fitted to the data by means of a KDF computer, on a program already available written by David Williams. Subject only to the condition that the two segments join, best least-squares fits are calculated successively for $z = 2$, $z = 2.01$, $z = 2.02$ etc. up to the maximum value of x . The best overall least squares fit is then chosen, thus determining the value of the 'turning point', z .

Three parameters are thus estimated for each series of responses: the slopes of the two linear segments and the 'turning point' where the two segments join, besides the two constants ' b ' and ' d '.

The prediction made is that if habituation is occurring, the slope of the first segment of the regression will be negative; if habituation does not occur it will be zero (or positive).

It is then possible to calculate whether the overall fit is significantly different from the null hypothesis that response magnitude is independent of trial number. This is done by an F test, comparing the variance accounted for by the regression with the residual sum of squares. (The residual sum of squares is the sum of squared differences between observed and fitted values).

The variance accounted for by the regression ("sum of squares due to regression") is the difference between the sum of squares (SS) of observed values about the mean, and the residual sum of squares. If the regression is estimated on data from N trials, then there are N-1 degrees of freedom (DF) associated with the SS about the mean, 3 DF associated with the SS accounted for by regression, and N-4 associated with the residual SS. Then

$$F \text{ is equal to } \frac{\text{SS due to regression}}{3} \div \frac{\text{Residual SS}}{4}$$

with 3 and N-4 DF.

The significance level thus obtained indicated whether any observed decrement in response during a series of stimuli could reasonably be assumed to be habituation, or whether it was most likely to be a chance effect. Thus it is possible to say whether or not there was significant habituation for each separate series of responses.

The slope of the first segment can also be used to provide a measure of the rate of habituation; although no particular importance should be attached to the actual value of this "rate", it can be used for comparative purposes. The "turning point", it was hoped, could provide an estimate of how many trials it took for habituation to be substantially complete - it provides another possible measure of the "rate" of habituation.

On a priori grounds one might expect habituation to take an exponential course. Hence it would have been reasonable to fit exponentials to the data. This was not done because it seemed to the author at the time that the two segment regression was better/

/better suited to fitting series of responses of variable length whose asymptote was not expected to be zero. At least this form of regression would appear to provide as adequate a test of habituation as any other. Lader (1964), and Lader and Wing (1966 , p.73) suggest that habituation of electrodermal responses may consist of two stages, the first stage with rapid habituation lasting only a few trials, followed by a period of slower habituation. Further, inspection of Figs. 6 and 7a in the results is as suggestive of a two segment form as of any other course of habituation. It should be pointed out again that the purpose of these experiments was not to define the precise form habituation might take in sleep, but rather to make a comparison between sleep and daytime conditions, with a view to seeing whether habituation in sleep was possible at all.

The method used here does have something in common with the methods used by Lader (1964) and Lader and Wing (1964), in so much as both their method and this fitted separate regressions for individual sets of data, and calculated whether each regression was significantly different from the null hypothesis. Both their method and this also used the parameters of the regressions for further comparisons. The methods differ however in that Lader and Wing assumed an exponential course for habituation instead of the two segment linear course assumed here. They were therefore forced to use a 'criterion' in conjunction with their regressions, namely that habituation had occurred if three successive stimuli elicited no response. Such criteria are frequently in use; they are, however, unreliable for responses whose absolute frequency is initially low/

/low (e.g. EEG responses), and clearly impossible to use with responses such as HRRs which do not decline to zero owing to ever present spontaneous fluctuations.

(c) The "Law of initial value"

Mention needs to be made of why Wilder's "law of initial value" has not been discussed in the data analysis. Lacey (1956) has developed techniques for controlling for the effect of the initial value or prestimulus level of autonomic responses on the magnitude (and direction) of the response. Lacey argues for various reasons that removing the effect of prestimulus values on response measures which are measures of change, by calculating the regression of one on the other and 'subtracting out' the effect of prestimulus values, is not a worthwhile procedure. Response measures which are measures of change include all responses defined in terms of the difference between two levels. Lacey argues that corrections to take into account prestimulus levels should be performed exclusively on post-stimulus (or 'stress') levels of the autonomic variable. A 'stress level' for heart rate for instance would be the value of the heart rate at some particular time after the stimulus. Thus the HRR as defined here cannot be considered a 'stress level'. Lacey argues for the use of "autonomic lability scores" (ALS) which are derived from the prestimulus and post-stimulus levels of the variable. The ALS also involves a correlation between initial and stress levels of the variable for the total sample of subjects. Thus the ALS is designed to measure the response of one subject relative to the mean response of the whole sample of subjects.

Lacey's argument is thus that while autonomic response measures may be dependent on prestimulus levels (this he says is only true for a group of subjects - such correlations are not high within any individual), it is not to any advantage to try and remove this dependence when measures of change are used to define 'responses'.

While Hord, Johnson and Lubin (1964) reported that the 'law' of initial values does in fact apply to heart rate following stimuli, it can now become apparent why 'corrections' to the heart rate responses, for prestimulus level, were not employed in this study. First, the corrections developed by Lacey are designed to be used on measures of a post-stimulus level. At least for heart rate, previous literature had indicated that not a level, but a response (in this case diphasic) was the best indicator of the effects of stimuli. Second, Lacey argues that no advantage is to be gained from any other attempts to 'correct' such measures in any way for prestimulus levels. One must therefore accept that the response measures used may be dependent on initial levels, and keep this in mind when interpreting the results.

In fact, Berg et al (1971) report that initial level of heart rate only accounted for between 5 and 20% of the total variance of heart rate in their analyses.

Baust and Marbaise (1970) maintain that HRRs to clicks in sleep are independent of initial heart rate levels. Certainly one would predict from Lacey's 'homeostasis' interpretation of the law of initial values that the effects of prestimulus heart rate on the diphasic HRRs as defined in this study would be minimal: if the 'peak' component were reduced by a high prestimulus heart rate, then the/

/the 'trough' would be enhanced, and vice versa. Keefe and Johnson (1970) have actually reported that, in their study of HRRS in waking, the peak-trough HRR measure was "not significantly correlated with prestimulus level".

However, the analysis of variance on prestimulus heart rate already described in connection with the definition of the HRR (Tables 4 and 5), will serve, it is hoped, to demonstrate that since no significant changes in prestimulus heart rate were occurring over trials, any habituation detected cannot be put down as an 'artifact' of the law of initial values.

Since the skin potential was recorded bipolarly as a difference in potential between two points, no basal values of skin potential were available to check whether changes in basal levels were related to the development of habituation. The relationship between skin potential basal level and response magnitude has been mentioned already (Chapter 3). It would indeed seem that response magnitudes are affected by basal level, in that, for instance, significantly more positive waves occur as part of the SPR when basal potential is highly negative (Trehub et al, 1962¹). But Wilcott (1958²) found that there was no relation between a diphasic SPR measure and basal skin potential. Wilcott's results of 1964³ provide the explanation: the magnitude of positive and negative waves are affected to an equal degree by basal potential, so that a measure which is the sum of the magnitudes of positive and negative components will be independent of basal level.

¹Trehub, A., Tucker, I. and Cazavelan, J. (1962) Epidermal b-waves and changes in basal potentials of the skin. Amer. J. Psychol. 75 140-143.

²Wilcott, R.C. (1958) Correlation of skin resistance and skin potential. J. Comp. Physiol. Psychol. 51 691-696.

³Wilcott, R.C. (1964) The partial independence of skin potential and skin resistance from sweating. Psychophysiology 1, 55-66.

However, Germana (1968) would argue that use of measures uncorrected for prestimulus levels leads to the finding of rates of habituation which are 'exaggerated' over the (by implication) 'true' rates. At least this suggests caution in interpreting the actual values for habituation rates.

CHAPTER SEVEN

PROCEDURE

1. Instructions:

Subjects were instructed to abstain from alcohol, tea, coffee, and other drugs (nicotine excepted), on the days of the experiments, and no subject took any medication except aspirin throughout the series of experiments. It was considered necessary to exclude any drug effects as it has been shown for instance that cyclobarbitone, amphetamines and chlorpromazine all facilitate electrodermal response habituation, at least in awake subjects (Lader, 1964; Lader and Wing, 1966, p.76; Scholander, 1961b; Rothschild and Connors, 1970), and in a review of drug effects on conditioning generally, even caffeine was reported to affect extinction (Dureman, 1959).

Subjects were told merely that noises would be played to them in the night, and they were also informed of what recordings were being made from the electrodes attached to them. In the daytime controls the subjects were asked to 'sit quietly and try not to move around' in a lighted room and to 'keep your eyes open'; they were also told that 'noises will be played to you over a loudspeaker'.

Subjects were required to spend two nights in the laboratory to adapt to the electrodes and to sleeping in the laboratory before the experiments proper began, and before any stimuli were presented. Thus the subjects' sleep could be characterised as 'normal' and reasonably free from 'first night effects' (Rechtschaffen and Verdane, 1964; Agnew et al 1966) such as increased shifts to wakefulness or stage 1.

2. Apparatus:

Subjects slept in an air-conditioned bedroom and took their normal hours of sleep. Silver disc electrodes using Cambridge electrode jelly were fixed with adhesive tape in supraorbital and outer canthus positions for eye movement recording (EOG), and two electrodes in midline (between Fz and Cz, and between Pz and Oz positions of the international 10/20 system) provided a fronto-parietal EEG. Electrodes placed submentally gave a record of neck muscle tone, which has been shown to reach a minimum during REM sleep (Berger, 1961). Skin potential was measured between the thenar eminence and scrubbed volar forearm, using a 0.3 sec time constant, gain 10 mm per millivolt. All derivations were bipolar. EEG and EMG were recorded at a gain of 14 mm per 100 microvolts, EOG at a gain of 10 mm per 100 microvolts. Time constants were 0.3 sec for EEG and EOG, 0.03 sec for EMG. SPR, EMG, EOG and EEG were recorded at a paper speed of 15 mm/sec in all night recordings on a 14 channel Alvar Reega electroencephalograph. A fifteenth channel provided a stimulus marker. An electrocardiogram was fed via an EEG amplifier into a Devices instantaneous ratemeter which provided a beat by beat record of heart rate, at a paper speed of 1 mm/sec. Heart rate was recorded only in the stage of sleep under study on the given night.

3. Stimuli:

The 'noises' to which the volunteers were subjected during the night were 1000Hz, 1 sec prerecorded tones presented over a loudspeaker situated across the cubicle from the subject, producing 70 db at the subject's head. 70 db was found in preliminary trials to be an/

/an intensity sufficient to produce a response ~~when~~ in stage 4, without waking the subjects. The decision to use a constant intensity tone for all sleep stages rather than an intensity graduated to the arousal threshold was made because the object of the experiment was to compare habituation in sleep with habituation in daytime controls to the same stimulus.

These stimuli were presented under one of the six ISI conditions on any one night. The six conditions were 10, 20 and 30 second regular intervals, and irregular ISIs with means of either 10, 20 or 30 seconds.

4. Details of the stimulation procedure:

Stimuli were presented when the subject reached the appropriate stage of sleep, excepting that 'descending' stage 2 at the start of the night was ignored, as was the first REM period, since these stages usually do not last many minutes. Subjects were not permitted to hear the stimuli before going to sleep, in contrast with the procedure of Johnson and Lubin (1967), who started stimulation while the subjects were still awake and continued stimuli uninterruptedly thence all night. In this experiment, subjects received stimuli only during one sleep stage under one ISI condition on any one night.

Prior to actual stimulation, when the appropriate sleep stage had been reached, a 'control' period of 5 minutes was allowed during which time no stimuli were presented. This period was allowed so that an assessment of the occurrence of spontaneous "responses" and of spontaneous variability of the measures could be made.

This procedure was considered preferable to that employed by Johnson and Lubin - they assessed spontaneous variability in the intervals between stimuli by a method which has been criticised already. Apart from the fact that their control procedure seems defective, with the shorter ISIs used in this study, their procedure would have been impossible.

After this control period, stimuli were presented. The series of stimuli were stopped if the subject altered sleep stage, or after 50 stimuli ('trials') had been presented. In order to ensure that the series of responses was long enough to allow any habituation present to be detected, if a series was interrupted before 20 trials had been presented, a further series (preceded by a new control period) was presented that night, allowing an interval of at least half an hour between successive periods of stimulation. On five occasions series with less than 20 trials were used, after five attempts, when it was felt that the initial response level might be affected if too many series of stimuli were presented in one night. Information as to the number of series presented, number of series terminated etc. is set out in Table 3.

No "dishabituation" test was made at the end of a series of trials to verify whether habituation had occurred rather than effector or receptor fatigue. This was necessitated by the procedure preferred here of continuing stimulation until the subject changed sleep stage - any 'test tone' presented afterwards would not fall within the same sleep stage as the experimental trials. Secondly,/

/Secondly, there have been a number of reports recently that intramodal test stimuli do not reliably elicit responses previously habituated to the experimental stimuli (Sokolov and Paramonova, 1961 ; Williams, 1963 ; McDonald and Carpenter, 1966 ; Geer, 1969 ; Houck and Mefferd, 1969). Geer specifically showed that in wakefulness prior presentation of experimental stimuli reduced the ORs to test stimuli. McDonald was conducting a test of habituation in sleep like the present one, and he reported dishabituation on only one in five to one in eight occasions. McDonald remarks that they were not surprised by this finding, as on Thompson and Spencer's (1966) model, dishabituation is merely incomplete stimulus generalisation.

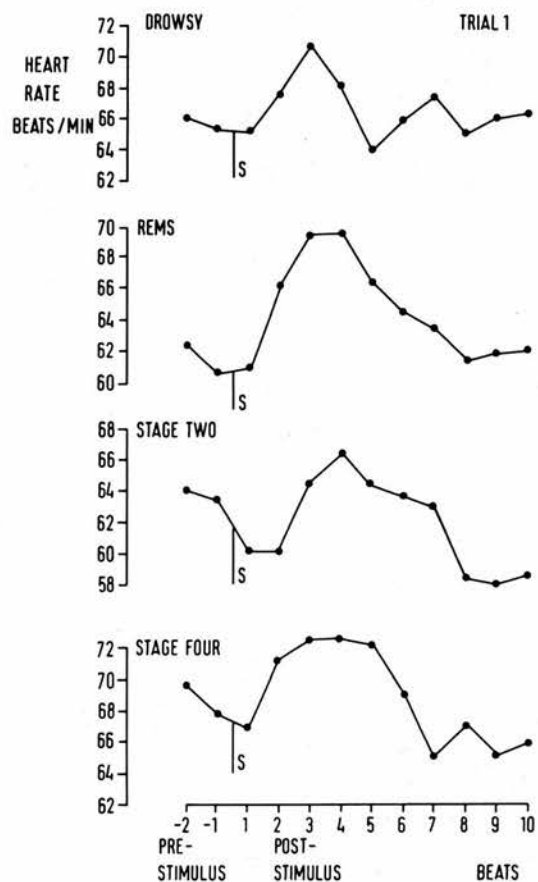
CHAPTER EIGHT

RESULTS

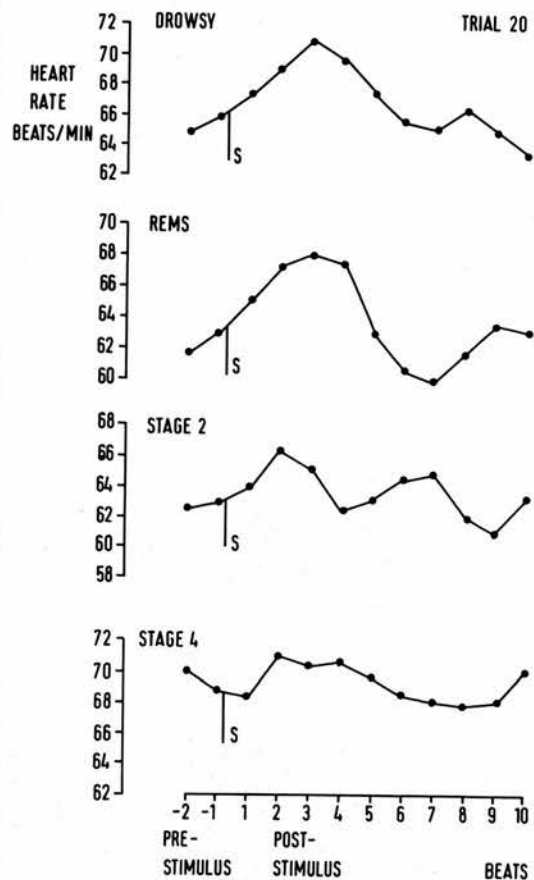
1. Summary:

Figures 6 and 7 illustrate the basic results for the two autonomic responses. Habituation of the skin potential response occurred in all states. Heart rate response habituation occurred in the two NREM sleep states, but it did not occur in the daytime controls (in a state of drowsiness), or in REM sleep. (There is in fact a suggestion that some habituation of the HRR is occurring in the first few trials in REM sleep, but the response becomes variable: there is no overall habituation). There was no effect of ISI on autonomic response habituation. However, sleep stage did appear to affect the rate of skin potential habituation, which was slower in NREM sleep stages (especially stage 4) than in REM sleep or in drowsiness (Figs. 8 and 9).

EEG responses showed habituation, both K-complexes in stage 2 and alpha responses in REM sleep (see Figs 10 and 11). K-complexes also showed a striking effect of interstimulus interval: habituation was rapid with short regular ISIs (10 sec), and almost non-existent with long irregular intervals of the order of 30 sec (see Fig. 12). This trend was present for the alpha responses also, but was not as marked (compare Figs. 13 and 14).



(a)



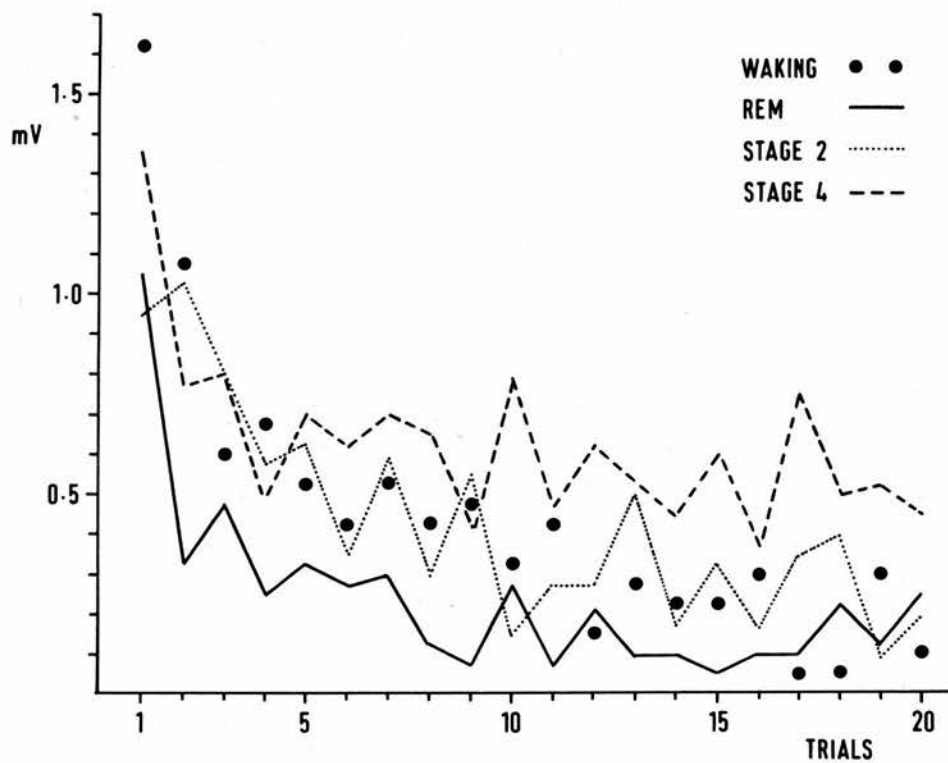
(b)

FIGURE 5. THE FORM OF THE EVOKED HEART RATE RESPONSE. MEANS FOR ALL INTERSTIMULUS INTERVAL CONDITIONS AND SUBJECTS ARE PLOTTED FOR:

- (a) Trial 1
- (b) Trial 20

Note how by trial 20 the response has almost entirely vanished in stage 2 and, especially, in stage 4. In REM sleep, however, it is still clearly present.

SKIN POTENTIAL RESPONSES



**FIGURE 6. MEAN SKIN POTENTIAL RESPONSE AS A FUNCTION OF TRIALS.
Habituation is unmistakable.**

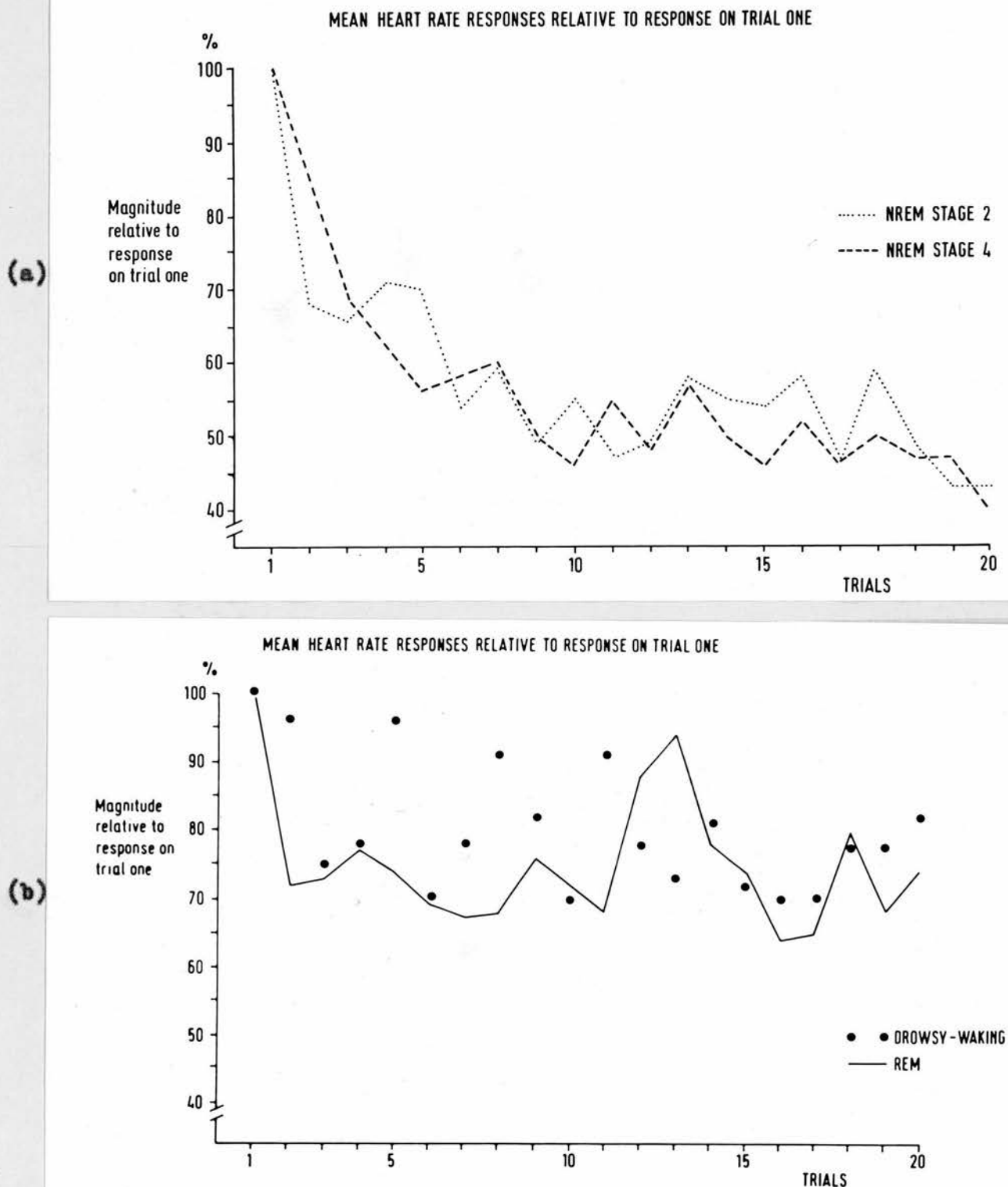


FIGURE 7. MEAN HEART RATE RESPONSE AS A FUNCTION OF TRIALS. TO EMPHASISE THE DIFFERENCES BETWEEN STATES, RESPONSES HAVE BEEN PLOTTED RELATIVE TO THE RESPONSE ON THE FIRST TRIAL.

(a) Non-REM sleep stages 2 and 4. Habituation is unmistakable.

(b) Drowsiness and REM sleep. Great variability in response magnitude, and little or no habituation.

"RATE" OF HABITUATION:
SLOPE OF FIRST SEGMENT OF REGRESSION
Means for all subjects and ISI conditions

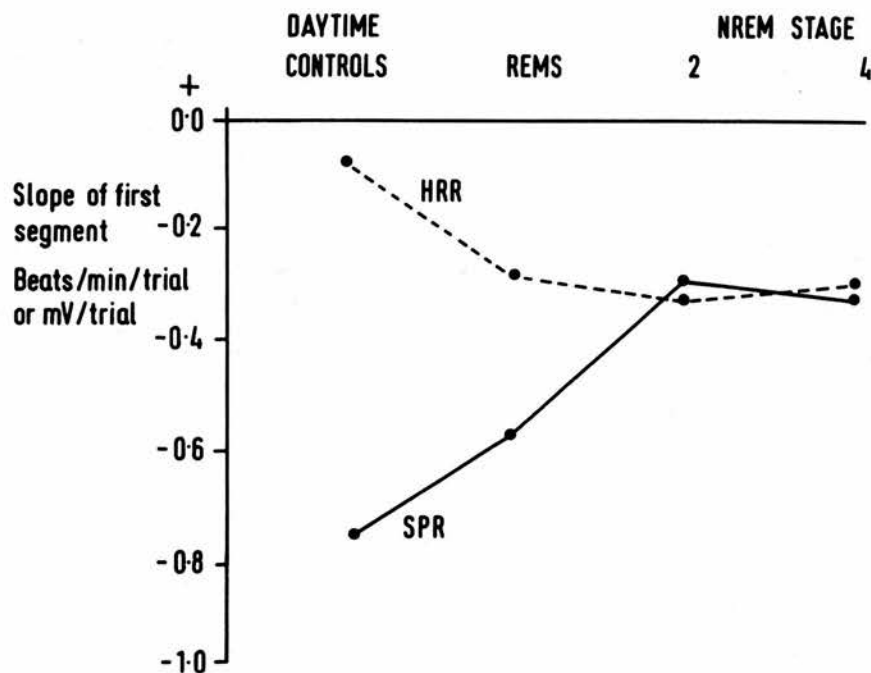


FIGURE 8. 'RATE' OF HABITUATION: SLOPE OF THE FIRST SEGMENT OF THE REGRESSION.

NUMBER OF TRIALS TO HABITUATION :
TURNING POINT OF REGRESSION
Means for all subjects and ISI conditions

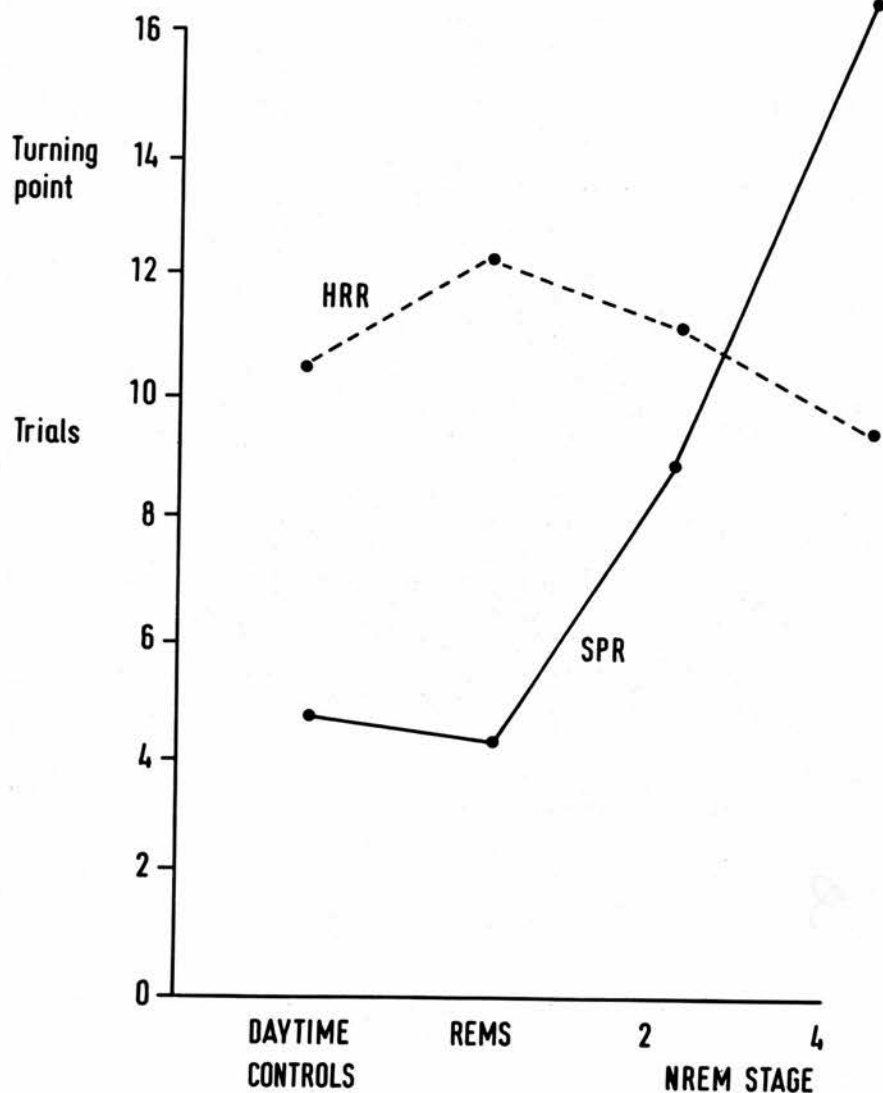


FIGURE 9. NUMBER OF TRIALS TO HABITUATION: 'TURNING POINT' OF THE REGRESSION.

The turning point of the regression can be interpreted as the number of trials before habituation is virtually complete. When the slope of the regression is zero or positive, however, as with the HRR in the daytime controls, then the turning point has little significance.

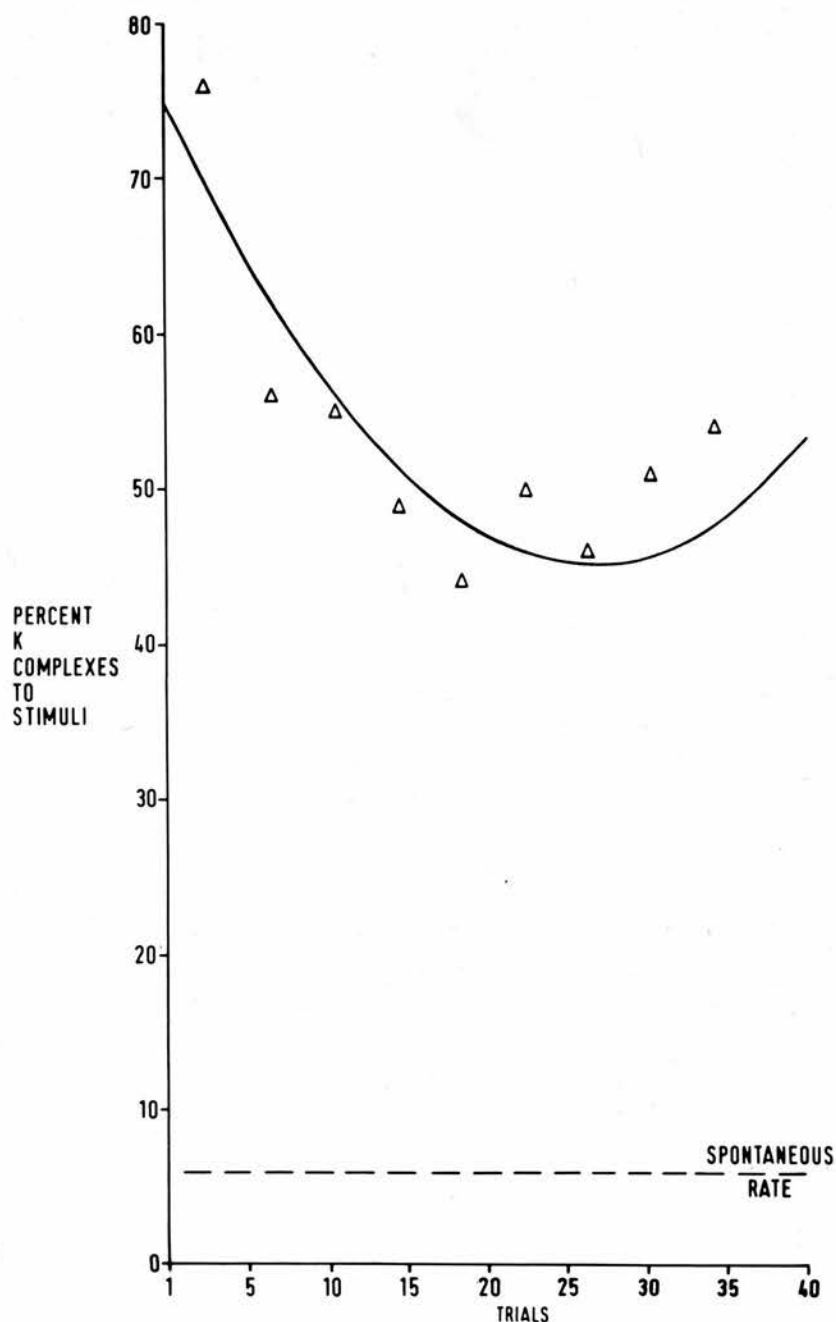


FIGURE 10. K-COMPLEX FREQUENCY AS A FUNCTION OF TRIALS. (STAGE 2 SLEEP).

Data are pooled for all interstimulus interval conditions, and plotted for blocks of four trials. The fitted curve is a best fitting polynomial. The 'spontaneous rate' indicates the frequency with which K-complexes are seen following the 'pseudo-stimuli' marked on the record.

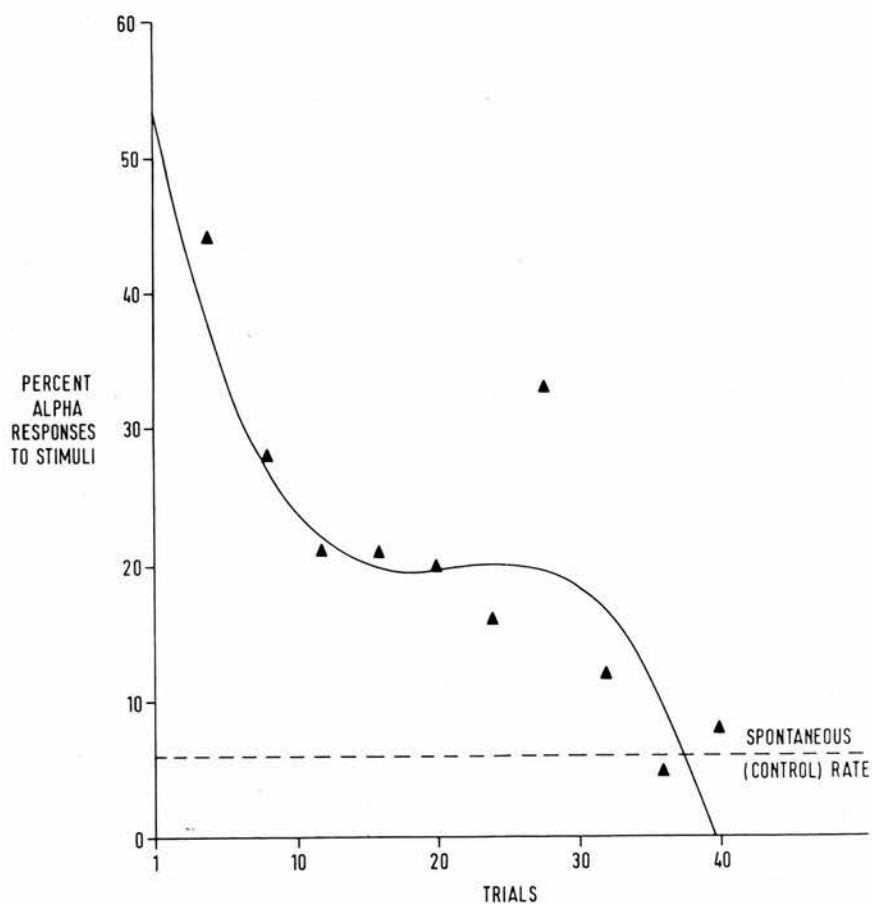


FIGURE 11. ALPHA RESPONSE FREQUENCY AS A FUNCTION OF TRIALS (REM SLEEP).

Data are pooled for all interstimulus interval conditions, and plotted for blocks of four trials. The fitted curve is a best fitting polynomial. The 'spontaneous rate' indicates the frequency with which bursts of alpha were seen following the 'pseudo-stimuli' marked on the record.

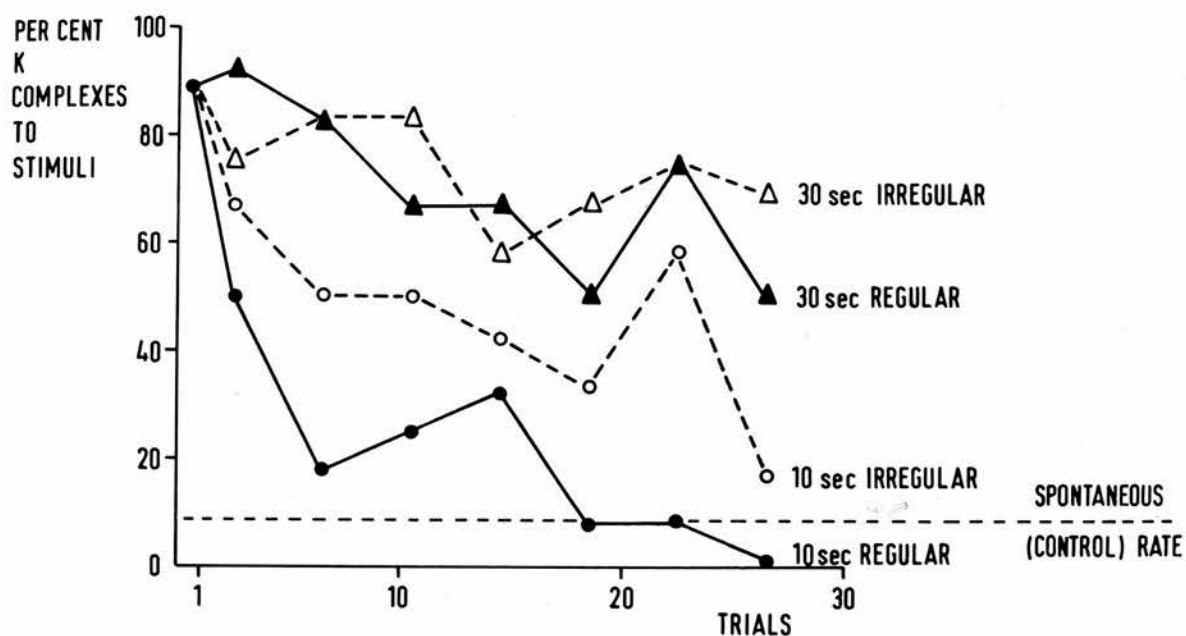


FIGURE 12. THE EFFECT OF INTERSTIMULUS INTERVAL ON THE HABITUATION OF K-COMPLEXES.

Response rates are plotted for four conditions. K-complexes occur to the first stimulus of a series on 90% of occasions. With short regular intervals between stimuli, habituation is rapid; with long irregular intervals habituation is almost non-existent.

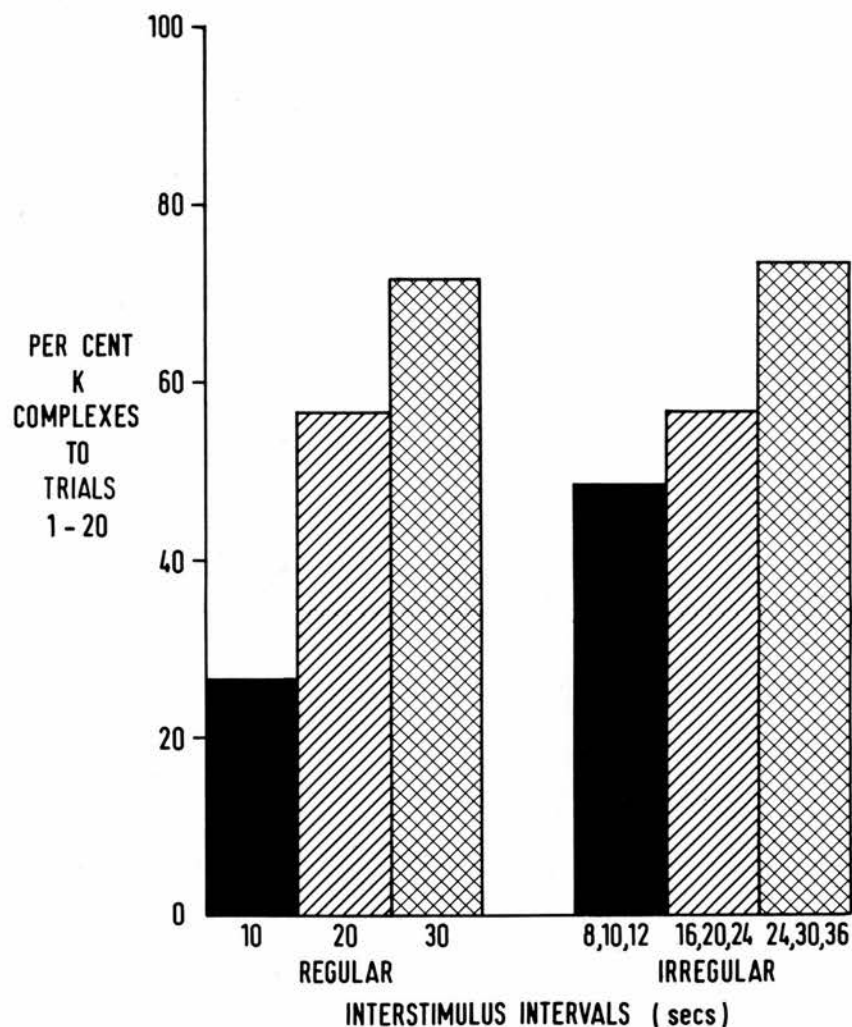


FIGURE 13. K-COMPLEXES OCCURRING DURING THE FIRST TWENTY TRIALS.

Rapid habituation makes the overall number of K-complexes low when intervals between stimuli are short and regular. With long and irregular intervals K-complexes continue to occur to most stimuli throughout the twenty trials.

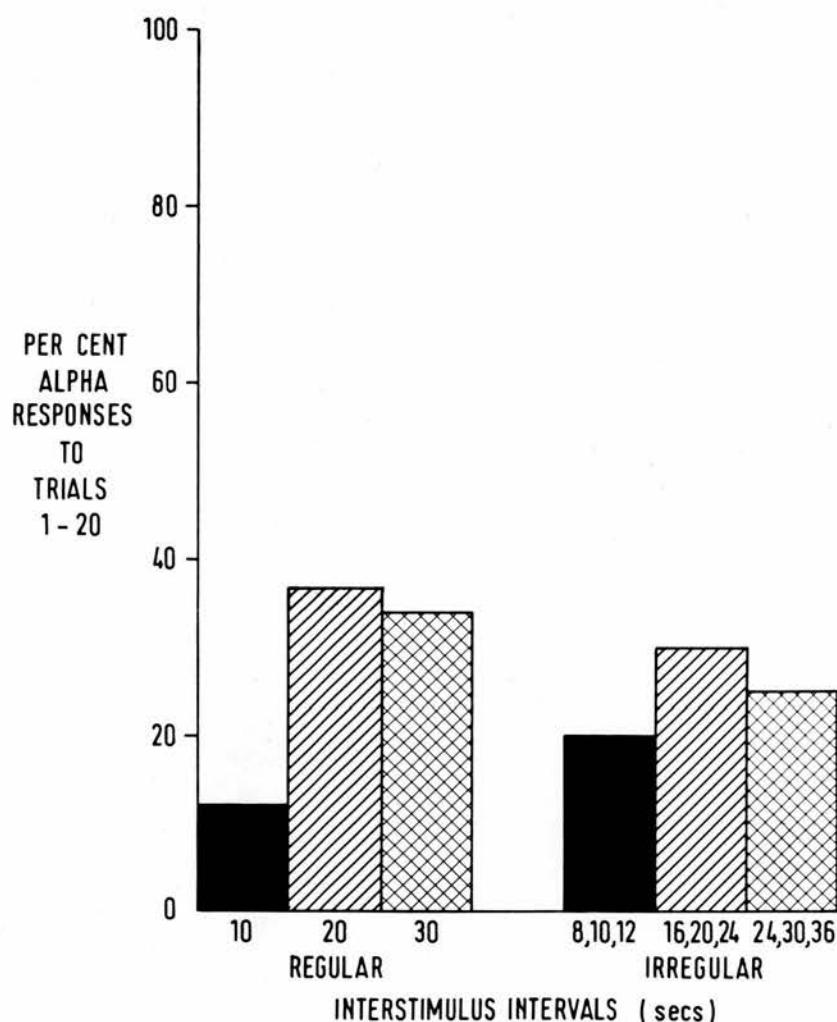


FIGURE 14. ALPHA RESPONSES OCCURRING DURING THE FIRST TWENTY TRIALS.

With short, regular intervals between stimuli alpha responses habituate rapidly; only a few occur in the course of twenty trials. With longer intervals between stimuli habituation proceeds more slowly.

TABLE 6.

SKIN POTENTIAL RESPONSES: SLOPES OF FIRST SEGMENT OF LINEAR REGRESSION. VALUES AND SIGNIFICANCE LEVELS ARE GIVEN FOR EACH OF THREE SUBJECTS IN EACH CONDITION (mv/trial).

STATE	ISI (sec)								
	REGULAR			IRREGULAR					
	10	20	30	10	20	30	10	20	30
Daytime Controls	-2.95 ***	-2.12 **	-0.26 *	0.00	+0.26	-1.20 ***			
	-0.25 ***	-0.79 *	-0.30 *	-0.12 **	-1.03 *	-0.12 *			
	-1.50 ***	-0.40 ***	-0.50 ***	-0.60	0.00	-1.50 ***			
REM	-1.70 ***	-0.02	-0.69	-0.79 *	-0.57	-0.80 ***			
	-0.40	-0.11 **	-0.07 *	-0.22 ***	-0.22	-0.60			
	-0.43 ***	-1.34	-1.01	-1.00 **	-0.46 ***	-0.10 ***			
2	-0.13	-0.27 ***	-0.80 *	-0.13 ***	+0.02	-0.39 ***			
	-1.01 ***	+0.03	-0.18 ***	-0.13 ***	-0.07 *	+0.12 ***			
	+0.06	-0.21 ***	-0.58 **	-0.02	-1.02 **	+0.02			
4	-0.03 ***	-1.51 ***	+0.08	-1.41 **	-1.00	-0.19			
	+0.01	-0.02	-0.07 *	-0.70 *	-0.05 *	-0.03 *			
	-0.56 *	+0.01 **	+0.01 *	-0.04	-0.60	-0.04			

* $p < .05$ ** $p < .01$ *** $p < .001$

TABLE 7.

HEART RATE RESPONSES: SLOPES OF FIRST SEGMENT OF LINEAR REGRESSION. VALUES AND SIGNIFICANCE LEVELS ARE GIVEN FOR EACH OF THREE SUBJECTS IN EACH CONDITION (BEATS/MIN/TRIAL).

STATE	ISI (sec)					
	REGULAR			IRREGULAR		
	10	20	30	10	20	30
Daytime Controls	-0.57	-0.75	-0.19	-0.30	+0.36	+0.45
	-4.83	-8.00 *	-5.20	* +0.06	+0.84	-2.26
	+0.22 *	+0.39	+0.24	-0.05	-0.31	-1.47 *
REM	-0.01	-0.24	+5.66	+0.09	-2.00	-0.55
	-12.42	-1.66	-4.00	* -10.42	-0.65	+2.25
	+0.27	-8.91	-8.40	-13.00	* +0.02 ***	-0.29 *
2	+0.04	-0.46 **	-1.71 ***	-16.53 ***	-4.00 *	-3.63 ***
	-7.00	+0.20	-0.46 *	-10.29 **	-15.00 ***	-0.21 ***
	+0.25	-0.46 *	-0.94 *	-0.17 *	-3.20 ***	-1.58 ***
4	-0.57 **	-14.51 ***	-0.84 **	-0.44 *	-0.12 *	-4.30
	-0.91 ***	-0.94 ***	-0.22	-8.17 ***	-2.07 ***	-7.00 **
	-0.27 *	-1.00	-5.00 ***	-0.90	-6.00 **	-1.16 **

* $p < .05$
 ** $p < .01$
 *** $p < .001$

TABLE 8.

SKIN POTENTIAL RESPONSE:Slopes of second segment of regression (mV / trial).Values are given for each of three subjects in each condition.

STATE	ISI (sec)					
	REGULAR			IRREGULAR		
	10	20	30	10	20	30
DAYTIME						
CONTROLS	-.004	-.008	.002	.000	-.013	.005
	-.006	-.053	.010	-.008	-.034	.270
	.003	-.003	-.006	-.003	.000	.000
REM	.001	.062	.028	-.002	-.006	-.004
	.006	-.009	-.001	-.008	-.039	.008
	-.005	-.011	-.068	.017	-.009	.000
NREM	.001	-.001	.016	.065	-.003	-.001
2	-.009	-.026	.000	-.016	-.004	-.027
	-.101	-.016	.006	.411	-.001	-.064
NREM	.095	.002	-.051	-.035	-.007	.007
4	-.011	.135	.189	.001	.026	.017
	-.004	-.031	-.019	.068	-.033	.056

Frequencies of negative and positive slopes are not significantly different in any state (Binomial test).

TABLE 9.

HEART RATE RESPONSES:

Slopes of second segment of regression. (Beats/min/trial).
Values are given for each of three subjects in each condition.

STATE	ISI (sec)					
	REGULAR			IRREGULAR		
	10	20	30	10	20	30
DAYTIME	.27	-.04	.46	.06	-.09	-.40
CONTROLS	-.07	.44	.21	-.22	-1.02	.40
	-.37	-.38	-.11	-.80	.88	.04
REM	-.60	-1.74	-.76	-.67	-.18	3.00
	.02	-.02	.96	.08	.14	-.07
	-3.00	.08	.43	.33	-.51	1.14
NREM	-.26	.04	-.21	-.06	.06	-.12
2	.01	-.15	.02	.06	-.32	-2.69
	-.11	.09	.75	-1.18	-.10	-.21
NREM	.01	-.01	.04	-.02	1.00	-.04
4	-.01	-.04	.30	.01	-.01	.01
	.17	-.07	-.04	-.03	.06	-.10

Frequencies of negative and positive slopes are
 not significantly different in any state (Binomial test).

TABLE 10.

SKIN POTENTIAL RESPONSES:Turning points of fitted regressions (trial number).Values are given for each of three subjects in each condition.

STATE	ISI (sec)					
	REGULAR			IRREGULAR		
	10	20	30	10	20	30
DAYTIME	2.0	2.0	6.8	(0.0)	5.0	2.0
CONTROLS	10.0	4.0	3.0	15.0	2.0	18.0
	3.0	2.6	6.3	2.5	(0.0)	2.0
REM	2.2	26.5	2.0	2.0	2.0	2.5
	2.2	4.2	4.0	9.9	3.0	2.3
	2.0	2.0	2.0	2.1	4.0	2.0
NREM	4.0	4.3	3.4	20.6	5.0	5.6
2	2.0	13.0	9.0	10.7	4.2	4.3
	12.9	10.0	3.0	28.0	2.0	20.0
NREM	38.0	2.0	4.3	2.0	2.0	5.0
4	35.0	32.0	21.0	2.9	30.0	11.0
	2.0	31.0	43.0	30.0	2.5	10.0

TABLE 11.

HEART RATE RESPONSES:Turning points of fitted regressions (trial number).Values are given for each of three subjects in each condition.

STATE	ISI (sec)					
	REGULAR			IRREGULAR		
	10	20	30	10	20	30
DAYTIME	9.0	6.0	8.0	12.0	12.0	10.6
CONTROLS	2.0	2.1	4.0	27.0	8.0	6.0
	9.0	9.0	25.0	16.0	15.0	6.0
REM	44.0	15.0	3.0	22.0	3.9	16.3
	2.0	7.0	5.7	2.0	17.0	6.0
	18.9	2.0	2.0	2.1	26.0	38.0
NREM	9.0	10.1	9.0	2.0	3.2	6.3
2	2.1	15.0	34.0	3.0	2.4	32.0
	4.3	24.0	10.0	25.0	6.8	8.0
NREM	10.0	2.0	7.0	10.0	35.4	2.0
	6.4	6.0	20.4	2.0	8.5	2.0
	23.6	4.1	4.2	5.8	2.9	6.0

TABLE 12.

SKIN POTENTIAL RESPONSE:Frequency of initial response decrement

STATE	Response decrement (negative slope of first segment of regression)	Positive slope of first segment of regression	Significance (Binomial test)
DAYTIME CONTROLS	15	1	$p < .001$
REM	18	0	$p < .001$
NREM 2	13	5	$p < .05$
NREM 4	14	4	$p < .02$

HEART RATE RESPONSE:Frequency of initial response decrement

STATE	Response decrement (negative slope of first segment of regression)	Positive slope of first segment of regression	Significance (Binomial test)
DAYTIME CONTROLS	11	7	ns
REM	13	5	$p < .05$
NREM 2	15	3	$p < .01$
NREM 4	18	0	$p < .001$

TABLE 13.

DAYTIME CONTROLS: EEG STATE AND HEART RATE RESPONSE HABITUATION

<u>Subject</u>	ISI					
	REGULAR			IRREGULAR		
	10	20	30	10	20	30
1	-.57	-.75	-.19	-.30	+.36	+.45
	1	1	0	0	1	0
2	-4.83	-8.00	-5.20	+.06	+.84	-2.26
	0	1	1	0	0	0
3	+.22	+.39	+.24	-.05	-.31	-1.47
	1	1	1	1	1	1

Slopes of the fitted regression (b/min/trial) are given together with an EEG 'sleep index'. A sleep index of 0 indicates less than 20 sec of stage 1 sleep in the experimental session: a sleep index of 1 indicates 20 sec or more of stage 1 sleep during the session.

TABLE 14.

SKIN POTENTIAL RESPONSES:SLOPE OF THE FIRST SEGMENT OF REGRESSION:Summary of analysis of variance.

SOURCE	SS	DF	MS	F	p
Subjects	2.200	2	1.100	8.10	.01
ISIs	1.247	5	0.249	1.49	ns
Subjects x ISIs	1.360	10	0.136		
States	2.660	3	0.886	6.16	.05
Subjects x States	0.863	6	0.143		
States x ISIs	5.306	15	0.353	< 1	ns
Subjects x States x ISIs	11.219	30	0.373		
Total	24.858	71			

TABLE 15.

HEART RATE RESPONSES:SLOPE OF FIRST SEGMENT OF REGRESSION:Summary of analysis of variance.

SOURCE	SS	DF	MS	F	p
Subjects	70.11	2	35.05	2.80	ns
ISIs	92.92	5	18.58	1.48	ns
Subjects x ISIs	125.48	10	12.54		
States	60.13	3	20.04	< 1	ns
Subjects	147.43	6	24.57		
States x ISIs	293.34	15	19.55	1.04	ns
Subjects x States x ISIs	563.84	30	18.79		
Total	1353.29	71			

TABLE 16.

SKIN POTENTIAL RESPONSES:TURNING POINT OF REGRESSION: (log transform)Summary of analysis of variance.

SOURCE	SS	DF	MS	F	p
Subjects	3.447	2	1.723	5.54	.05
ISIs	2.751	5	0.550	1.76	ns
Subjects x ISIs	3.112	10	0.311		
States	12.390	3	4.130	5.03	.05
Subjects x States	4.931	6	0.821		
States x ISIs	6.986	15	0.465	< 1	ns
Subjects x States x ISIs	22.985	30	0.766		
Total	56.604				

TABLE 17.

HEART RATE RESPONSES:TURNING POINT OF REGRESSION: (log transform)Summary of analysis of variance.

SOURCE	SS	DF	MS	F	p
Subjects	1.458	2	0.729	1.47	ns
ISIs	0.455	5	0.093	< 1	ns
Subjects x ISIs	4.942	10	0.494		
States	0.801	3	0.267	< 1	ns
Subjects x States	2.735	6	0.455		
States x ISIs	14.501	15	0.966	1.57	ns
Subjects x States x ISIs	18.451	30	0.615		
Total	43.357	71			

TABLE 18.

SPONTANEOUS AUTONOMIC ACTIVITY:

'Responses' to pseudo-stimuli. Means and standard deviations for each subject in each state. Means are for 10 'responses' for each condition, i.e. for 60 responses in each state.

Skin potential: (mV)

STATE	Mean for each subject	s.d.	Mean for all subjects ¹	s.d.	Mean response evoked by stimulus trial 1.
Daytime	.09	.40	.12 mV	.35	1.63 mV
Controls	.25 .06	.44 .12			
REM	.18 .22 .12	.28 .40 .37	.17 mV	.35	1.06 mV
NREM	.08	.12			
2	.07 .27	.19 .39	.14 mV	.26	0.93 mV
NREM	.15	.65			
4	.29 .35	.53 .67	.26 mV	.63	1.36 mV

Heart rate responses: (beats per min)

Daytime	4.33	1.59			
Controls	3.04 5.72	2.05 2.25	4.36 beats/min	1.98	6.80 beats/min
REM	3.90 3.43 3.60	2.28 2.54 2.14	3.64 Beats/min	2.39	7.85 beats/min
NREM	2.33	1.28			
2	1.74 3.35	1.15 1.83	2.48 Beats/min	1.47	8.10 beats/min
NREM	2.65	1.46			
4	1.34 3.65	1.01 1.51	2.55 Beats/min	1.36	7.30 beats/min

¹ sd for group calculated from Oswald et al (1971). $sd = \sqrt{\frac{\sum (N_i \cdot sd_i^2)}{\sum N_i - J}}$ where N_i = No. obsns. for each subj, where sd_i is derived, J = Total No.

2. Autonomic responses:

Figures 6, 7, 8 and 9 illustrate the results of the experiments for autonomic responses. Tables 6 to 9 show the slopes of the two segments, and Tables 10 and 11 the turning points of the regressions fitted to each series of responses. Tables 6 and 7 also show whether or not the fitted regressions were significantly different from the null hypothesis that response magnitude was independent of trial number. Significant skin potential response decrement is widespread in all states; the HRR shows significant response decrement frequently in both the NREM sleep states, but very rarely in the daytime controls or in REM sleep. The contrast for the HRR between NREM sleep states and REM sleep is striking if Figures 7a and 7b are compared. Examination of Figure 5, illustrating the magnitude of the HRR at trials 1 and 20, provides further evidence of HRR habituation in the NREM sleep states.

The Daytime controls: The daytime conditions were intended to provide a 'control' study of habituation in wakefulness. It was naturally expected that both the HRR and the SPR would habituate rapidly in these daytime control conditions. However, as can be seen from Table 7, the HRR only rarely showed significant habituation. The reason for this became clear when the daytime records were re-examined. Subjects had been drowsy during the daytime sessions. Five of the daytime sessions were abandoned at the time, and re-run at a later date, because subjects fell asleep: K-complexes or spindles appeared. Although these sessions were totally discarded, it is not surprising that in the remaining sessions the control conditions tended to/

/to make the subjects drowsy - a warm room, monotonous stimulation and instructions to sit quiet. A re-examination of the EEGs in the daytime controls showed that subjects were indeed drowsy (20 seconds or more of EEG stage 1) in half of the daytime sessions (see Table 13).

The HRR in REM sleep: Despite the lack of significant habituation of the HRR in REM sleep (Table 7), there is evidence that some habituation of the HRR is taking place in REM sleep, for negative slopes in the first segment of the regression (response decrements) are much more frequent than positive slopes in both REM and NREM sleep - though not in the daytime controls (see Table 12). Sign tests reveal the frequency of response decrement to be significant ($p < .05$) for the HRR in NREM stages 2 and 4 and in REM sleep. (Such differences are significant for the SPR in all states).

The "turning point" of the regressions: SPR and HRR: Examination of the fitted slopes of the second linear segments (beyond the turning point of the regression) in Tables 8 and 9 shows that positive and negative slopes are equally common. Their frequency is not significantly different for either of the two autonomic measures in any of the four states (Sign test, Siegel, 1956). Thus it does seem plausible to interpret the turning point as an approximate measure of the number of trials habituation takes; beyond that point responsiveness has reached a fairly stable level.

3. Differences between interstimulus intervals and between states in autonomic response habituation:

Analysis of variance for the slopes of the first segment/

/segment and for the turning points of the regressions were used to see whether ISI or sleep stage was affecting these measures of 'rate' of habituation. Summaries of the analyses are set out in Tables 14 to 17. There are no significant effects of inter-stimulus interval.

(a) Skin potential responses: For SPRs, there are significant effects of state (or sleep stage) for both the slope ('rate' of habituation) and for turning point (number of trials to habituation). However, although the F test shows significant effects ($p < .05$), orthogonal comparisons following the analysis, between pairs of states, were unable to reveal where these differences lay. It would seem therefore wise to conclude that differences between states in 'rate' of SPR habituation approach significance. Figures 8 and 9 show the mean slopes and turning points, for each state, for the SDR and the HRR.

(b) Heart rate responses: Regarding the HRR, a seeming paradox has emerged. In the drowsy daytime controls and in REM sleep, only on a very few occasions did the overall regressions indicate significant habituation, in contrast to frequent habituation in the NREM sleep stages (Table 7). However, analysis of variance on the "rate" of habituation reveals no significant differences between the NREM states, and REM sleep and drowsiness.

A look at the graph of mean response versus trial number (Fig. 7) may help to elucidate this, at least for the HRR in REM sleep. In REM sleep the HRR declines sharply in the first few trials - as/

/as mentioned earlier, in REM sleep a significantly larger number of the fitted regressions show an initial fall in response than show a rise (Table 12). But the HRR becomes very variable: in contrast to NREM sleep, the HRR in REM sleep remains high and it fluctuates. It would appear that the regression picks up the initial fall in response in the first few trials, but because this decline is not sustained, the fitted values for all trials do not differ significantly from the null hypothesis of a maintained response. Thus we can conclude that some degree of HRR habituation does occur in REM sleep, at least in the early trials, but it becomes irregular, and variability of the HRR is large among the later trials. It is of interest that the evoked HRR should show high variability in REM sleep, as spontaneous variability of heart rate in the absence of stimuli is also high in REM sleep. Spontaneous variability was assessed by scoring the 'response' to 10 'pseudostimuli' marked in the control period preceding each series of stimuli. Means and standard deviations of these spontaneous 'responses' are given in Table 18.

In the drowsy state, although the mean slope of the regressions is very slightly negative, negative and positive slopes are equally common.

We must therefore conclude that in drowsiness the HRR does not habituate while in REM sleep habituation is irregular and inconsistent: habituation in the early trials is not maintained and responses over all trials do not differ significantly from responses on the first/

/first trial.

4. EEG responses: K complexes in stage 2 and alpha responses in REM sleep:

Figures 10 and 11 show the mean response rates for the EEG responses over trials 1 to 40. In these figures responses from all subjects and ISIs have been combined and plotted for blocks of four trials (polynomial curves were also fitted for these figures).

For the K-complexes habituation is very evident in the first ten to twenty trials, though its course appears uncertain beyond trial 25 (until the effect of ISI is examined, when it will become clear).

Habituation of alpha responses is also striking in the first twenty trials. Though there is considerable variation in response rates after twenty to thirty trials, habituation becomes complete (i.e. to spontaneous levels) within 40 trials.

Effect of interstimulus interval:

The fitted curves in Figures 10 and 11 combine the data for all ISI conditions. Thus it would seem from Figure 10 that K-complexes do not habituate completely, but continue to occur to about 50% of stimuli - roughly half their frequency on trial 1. However, a breakdown by ISI condition shows that with the short ISIs habituation was complete, while with the longer ISIs it was very slight.

A fairly crude but simple way of looking at the effect of ISI is illustrated in Figs. 13 and 14. These show the mean response rate over the first 20 trials. Where habituation is rapid the overall response rate is low. Thus it can be seen from Figs. 13 and 14/

/14 that habituation is occurring more rapidly with the 10 sec ISIs than with the longer intervals. For example, with a regular 10 sec interval, alpha responses occurred to roughly 10% of stimuli in the first twenty trials, while with longer intervals responses occurred to roughly 30-40% of stimuli.

For K-complexes it was possible to plot the frequency of responses for each of the ISIs (Fig. 12), and again it is clear that there is a dramatic effect of ISI. Habituation is fastest with the regular 10 sec ISI, slower with the irregular short interval, and very slow with the two long ISI conditions. (The intermediate ISI conditions are omitted from the figure for clarity).

Thus habituation of K-complexes was more rapid with short ISIs than with longer ISIs. Also, habituation took place more rapidly with a regular interval than with an irregular interval of the same mean duration. The frequency of alpha responses was too low to justify plotting separate curves for each ISI.

In the control period before stimulation, the rate of occurrence of K-complexes, and also of alpha responses at 'pseudostimuli' was found to be 6%. It is striking that with a regular 10 sec ISI the frequency of K-complexes had habituated after only 20 trials to this 'spontaneous' rate. By contrast, with long irregular stimulus intervals responses were still occurring to 70% of stimuli after thirty trials.

CHAPTER NINE

DISCUSSION

The major conclusion to be drawn from the results described above is that during sleep there is at least one autonomic response, the skin potential response, which will consistently wane or habituate on repeated presentation of an auditory stimulus in all stages of sleep. The other autonomic response studied, the heart rate response, will reliably habituate in NREM stages of sleep, but does not do so either in REM sleep or in drowsiness.

1. Initial response magnitude and autonomic response habituation

The results can first be discussed with reference to the second section of Chapter 3. Before discussing the evidence about habituation, first we should note that both the heart rate response and also the skin potential response were elicited in all stages of sleep.

Thus the suggestion of Keefe et al (1971) that SPRs cannot be elicited from sleep without waking the subject is plainly not supported by these results: it is possible to elicit the SPR in all stages of sleep. It was stated in Chapter 3 that electrodermal activity was generally lowest in REM sleep. Here, initial SPRs were in fact least in stage 2 sleep, not REM sleep. But a glance at Figure 6 shows that except for the very first trial, responsiveness was lower in REM than in other stages of sleep. Thus these results do not in fact suggest a pattern of electrodermal responding which is different from that described in Chapter 3.

The heart rate responses found on trial one (Figure 5) were of essentially the same form as those described by Hord et al (1966). The magnitude of the HRR was greatest not in REM but in NREM stage 2 in this study. Although Hord et al emphasise the magnitude of responses in REM sleep, they too in fact found marginally greater responses in stage 2 than in REM sleep.

Habituation and initial response magnitude:

Lader (1964) and Lader and Wing (1964) found correlations of -0.68 and -0.81 between the magnitude of the slope and the intercept of their regressions (of response magnitude on stimulus number). They interpreted this as indicating that rates of habituation were correlated with initial response magnitudes, and "corrected" their rates of habituation for "initial values". It might be wondered what was the relation of initial response magnitudes to habituation the results here. Before that is done, however, it is necessary to make a couple of points about the procedures used by Lader and Wing (1964). The first issue is of general importance. Lader found a significant correlation between two values, and "accordingly" corrected one value to remove its correlation with the other. This is a procedure readily used in psychophysiology but one which has dangers. If rates of habituation are related to initial response, this is an interesting association, but it does not follow that the measure of 'rate' is not most useful as it stands. In Lader and Wing's study, most information is in fact conveyed by showing that normals have initially large responses which habituate rapidly, whereas their patients had small responses which habituated slowly. If the effect of initial response on habituation rate is removed, then much/

/much of this information is lost. For this reason no attempt will be made here to "correct" the 'rates' of habituation for initial response magnitudes.

The second point which needs mentioning about Lader and Wing's procedure is that they did not genuinely relate rates of habituation to initial responses. They used instead the intercept from their fitted regressions. These regressions specifically excluded the initial response. Lader and Wing in fact showed that there was no significant difference between the magnitude of the first response in the normals and the patients (1964, Table II). Yet they then go on, on the same page (p.214), to say that their normal subjects have larger "early" responses. They can only come to a conclusion so at variance with their data because of their strange procedure of actually disregarding the response to the first stimulus in all their statistical treatments.

Lader (1964) claimed to have found that GSRs habituated exponentially. That is, the responses had been successfully fitted to the logarithm of the stimulus number. But this had only been achieved by ignoring the initial response (which is plotted separately in all these workers' graphs). Lader's only justification for this was that "the first response is distinct"! Lader also commented that the last responses in the series did not fit an exponential either, but rather "the last responses do not show any tendency to decrease and the line may in reality have a point of inflection toward the end and flatten out". (1964, p.327). Lader and Wing's procedure also necessitates disregarding responses after/

/after three consecutive failures to respond (1966, p.68).

For these reasons Lader and Wing's techniques are not preferred here. A correlation has however been calculated for each of the autonomic responses, to express the relation between the magnitude of the first response and the slope of the first segment of the regression.

These correlations are given below:

For skin potential responses, $r = - .47$

For heart rate responses, $r = -.42$, when results for all states are combined. The correlations for each individual state are as follows:

Skin potential response, daytime controls,	$r = - .47$
REM sleep,	$r = - .35$
NREM stage 2,	$r = - .39$
NREM stage 4,	$r = - .39$
Heart rate responses, daytime controls,	$r = - .30$
REM sleep,	$r = - .51$
NREM stage 2,	$r = - .45$
NREM stage 4,	$r = - .51$

Over all states, correlations are significant for both autonomic responses; but within states, only correlations of .46 or more are significant.

Thus we can see that the magnitude of correlations between initial response and rate of habituation is much less than those found by Lader (1964) and Lader and Wing (1964). Indeed the correlations are only consistently significant when data from all states are pooled. This suggests that habituation rates may be greater in states where the initial response is greater. A comparison of Figure 8 with the mean responses to trial 1 (Table 18) confirms that this is indeed true. Thus for SPR, largest initial responses occur in the daytime controls, and this is the state showing the greatest habituation rate. For HRR,/

/HRR, daytime controls manifest the smallest initial responses, and it is in this state that habituation is least evident. Interestingly, it is in the daytime controls for the HRR that the lowest correlation exists between initial response magnitude and slope of the first segment of the regression. The noteworthy point about these results, however, is the relatively small magnitude of these correlations compared with those reported by Lader and Wing; at the most, only 25% of the variance¹ in habituation rates was related to the initial response magnitude.

It might have been expected that initial evoked response magnitudes would be related to the prevailing magnitude of spontaneous activity in the various states. In fact this relation is not borne out by these data: SPR initial response magnitudes are highest in the daytime controls, whereas it is in NREM stage 4 sleep that spontaneous electrodermal activity has been described as being greatest (see Chapter 3). Similarly, the HRR to the first trial was not greatest in REM sleep. The spontaneous activity recorded in this study, given in Table 18, shows quite clearly that there is little relation between initial response magnitudes and spontaneous response magnitudes when comparisons are made across states, either for skin potential responses or for heart rate responses. There is no relation between the rank order of states for spontaneous activity, and the rank order for initial response magnitude.

¹The variance in one variable accounted for by another variable equals the square of the correlation coefficient between them.

2. Habituation of the heart rate response

(a) The daytime controls:

In retrospect, that subjects became drowsy in the daytime controls is hardly surprising. Oswald (1962) reviewed the evidence that monotonous stimulation could induce sleep, including his own experiments (Oswald, 1959, 1960), and he concluded that repetitive, even noxious stimuli could induce sleep in some subjects. Both Pavlov (1927) and Sokolov (1963a) also accept that repetitive stimulation can lead to the onset of sleep. Although Tizard (1966a) concluded from her experiments that monotonous stimulation is no more efficient than an absence of stimulation in inducing sleep, Bohlin (1971) has recently criticised Tizard, in particular for not allowing subjects sufficient time for the soporific effects to work. Tizard's stimuli lasted only 8.5 minutes; the time taken for most subjects to fall asleep at night is of the order of 20 minutes. Bohlin found that without stimulation, mean sleep onset time by day was nearly 30 minutes; with an 80 db tone presented at an irregular interval averaging 30 sec, sleep onset time was shortened to 20 minutes. She specifically associated the development of sleep with the habituation of the OR. In the present study there was no evidence to support this (Table 13) - though clearly this study was not designed to test Bohlin's hypotheses.

Granted that these subjects did in fact frequently become drowsy, then the lack of habituation of the heart rate response is in agreement with previous work. Besides the descriptive reports of Sokolov (1963a) and Sokolov and Paramonova (1961) that habituation may be peculiarly/

/peculiarly difficult in drowsiness, McDonald et al (1964) examined both cardiovascular and electrodermal responses in alert and drowsy subjects. They found that while the GSR habituated reliably in both groups of subjects (as did the SPR in this study) heart rate responses did not habituate in the drowsy group. However, unlike McDonald et al's study, there was no consistent relationship in this study between the EEG status of subjects and whether or not the HRR habituated (Table 13). The EEG criteria of drowsiness were essentially the same in both studies: a subject was classified as 'drowsy' if he showed signs of drowsiness at some time during the presentation of stimuli.

(b) The heart rate response in REM sleep:

Since it is already known that the HRR will not habituate in drowsiness (McDonald et al 1964), the most interesting finding of this study is the lack of significant habituation of the HRR beyond the first few trials in REM sleep.

Before going on to discuss this, however, it is necessary to consider whether this unexpected result may not have been the result of artifact. In Chapter 3 various factors controlling or affecting heart rate were considered. It is now pertinent to ask whether any of these factors could have been responsible for the finding of no HRR habituation in REM sleep.

As was discussed in Chapter 3, temperature and cerebral blood flow and cerebral oxygen consumption are different in different stages of sleep. These factors may influence heart rate, and indeed heart/

/heart rate is generally different in REM and NREM sleep.

Moreover, rectal temperature, respiration rate, and oxygen consumption vary during the night (Snyder, 1971), and there is a time of night effect in the magnitude of autonomic responses (Williams et al 1964; Johnson and Lubin, 1967). However, changes in response magnitude during the night will not affect the magnitude of responses over the course of one series of trials, lasting at the most twenty-five minutes.

Possible artifacts on the HRR in REM sleep are therefore confined to phasic changes in the various factors affecting heart rate during REM sleep. In Chapter 3 it was concluded from the studies of Aserinsky (1965b¹) and Spreng et al (1968) that the phasic alterations in arterial oxygen which occur in conjunction with bursts of eye movements, are not in fact associated with fluctuations in heart rate, surprising as this may sound. There are no descriptions as yet of phasic changes in cerebral blood flow, cerebral oxygen consumption or body temperature during REM sleep (as distinct from the literature on tonic changes during REM sleep). However, given the observations that phasic changes associated with eye movement bursts are not associated with changes in heart rate we can only presume that the cardiac homeostatic mechanisms may be sufficient to stabilise heart rate from these influences.

Over the period in which stimuli were applied during REM sleep (and indeed during all stages of sleep) there is thus no reason to suppose progressive influences during REMs on heart rate responses which would either accentuate or mask habituation. There is, though/

¹Aserinsky, E. (1965b) Periodic respiratory pattern occurring in conjunction with eye movements during sleep. Science 150 763-766.

/though, one feature of REM sleep which might be supposed to affect the detectibility of response decrement: the degree of variability in heart rate.

Respiration rate is more variable in REM sleep than in other sleep stages (Aserinsky, 1965; Hobson et al 1965). Sinus arrhythmia is accentuated in REM sleep (see Figure 3), and indeed Snyder et al (1964) found that minute to minute heart rate variability was greater in REM sleep than in NREM sleep. The likely result of this on measured heart rate responses in REM sleep is an increase in the variability of the measured HRRs. An increase in the variance of responses might mask habituation which was actually occurring. Figure 7b suggests that this may indeed have been occurring. However, the regressions used did detect an initial response decrement (Table 12) despite any such effect. (Incidentally, if this response decrement was largely confined to the first and second trials, as Figure 7b suggests, Lader and Wing's regression techniques (1964) would not have detected this). It remains a possibility to be borne in mind, that increased sinus arrhythmia was responsible for masking any further HRR habituation in REM sleep.

It is convenient at this point to discuss two other studies. The one previous published study which did find a lack of habituation in REM sleep was that of Martinus and Papousek (1970). They claimed that the eye-blink reflex failed to habituate in the REM sleep of babies. However, they provided no numerical information on this and since they used only visual means to evaluate the sleep stage, their finding/

/finding must be treated with a considerable degree of scepticism. The only other study looking at each sleep stage separately which found HRR habituation in NREM sleep also found HRR habituation in REM sleep (McDonald and Carpenter, 1966).

The differences in methodology between this study and McDonald and Carpenter's are few. They used a 500Hz tone, of intensity only 40 db, whereas a 100 Hz 70 db tone was used here (they did choose the low intensity to maximise the chance of detecting habituation). They presented 30 stimuli in any one series of trials, with an ISI randomly varying between 10, 15 or 20 seconds. They allowed 30-40 minutes between series of trials, presenting stimuli on one night first in stage 4 sleep, then in REM sleep, then in stage 2, and again in REM sleep and stage 2. They also interpolated 'test trials' at frequencies of 200, 2000 or 4000 Hz, after the tenth and twentieth trials of each series. None of these differences in procedure would seem able to explain the fact that McDonald and Carpenter found consistent habituation of the HRR in all stages of sleep, while in this study differences between states were noticed.

However, there are differences in the analysis of the data. In their paper McDonald and Carpenter do not say how they assessed whether habituation was significant - all they say is that the HRR showed a "consistently significant decrease". Their report is still unpublished; McDonald has however explained in a personal communication that they compared the first two and the last two trials for each subject, using a sign test.

Now in the present experiment it will be recalled that in REM sleep there was a consistent decrease in the HRR between trials 1 and 2 (Fig. 7b), although over twenty or more trials this was only very rarely consistently maintained and so was only rarely significant. Since moreover there were no significant differences between states in 'rate' of habituation as judged by the slope of the regression over the first few trials, it appears that the differences between this study and that of McDonald and Carpenter are more apparent than real. Both studies found in REM sleep an initial decrement in the HRR between the first few trials and subsequent trials. In this study, however, this initial decrease in responsiveness was not maintained, but instead became irregular - or the decrease was masked by high variability of the heart rate; there was no overall significant habituation (Figure 3c illustrates this). The response even recovers to its magnitude on the first trial at times (Figure 7b).

(c) The similarity of HRR behaviour in drowsiness and REM sleep:

If the finding that the HRR in REM sleep would not habituate beyond the second trial is correct, and is not merely due to a masking effect of high heart rate variability, it is certainly unexpected. However, it is noteworthy that the EEG is to a large degree similar in stage 1 (drowsiness) and in REM sleep, despite their being totally distinct states physiologically. One is therefore tempted to conclude that the lack of HRR habituation in both drowsiness and REM sleep is related to their common level of/

/of EEG activation¹. Johnson et al (1970) have recently done a study in which waking, REM sleep and stage 1 on the one hand, and NREM stages 2, 3 and 4 on the other hand, were discriminated into two entirely separate groupings by means of a spectral analysis of the EEG. The waking EEG can be discriminated from that of stage 1, but stage 1 is not discriminable from REM sleep on the basis of the EEG alone - except when bursts of saw-tooth waves precede bursts of eye movements in REM sleep - (Lubin, Johnson and Austin, 1969 ; Johnson, Lubin, Naitoh, Nute and Austin, 1969 ; Johnson, 1970). However, since HRRs habituate in both alert, awake and in NREM sleep states, HRR habituation is clearly not a linear function of the level of cortical activation¹.

That habituation patterns are in some way dependent on the level of cortical activation might at first sight lend support to Sokolov's theory that habituation of OR components is largely a function of cortical inhibition. As mentioned in the introduction, Sokolov's model would predict that with increasing cortical synchronisation the inhibitory role of the cortex on the OR would be steadily diminished. Thus from wakefulness through drowsiness and REM sleep, NREM stages 2, 3 and 4 habituation of any response component would be expected to proceed more and more slowly. This would seem to/

¹The term 'activation' is used here to refer specifically to the EEG. It is not intended to imply any unitary dimension of activation, since it has been argued with justification that the concept of a unitary dimension of arousal or activation has little validity (e.g. Lacey, 1967). Increased EEG activation is here taken to mean decreased intensity of low frequency (0-4Hz) activity in the EEG.

but it is certainly not true of the HRR. Sokolov's theory therefore will not explain the curious behaviour of the HRR, habituating at high and low but not intermediate levels of cortical activation.

Eysenck's inhibition-excitation model of conditioning (1957), which assumes 'depression' of the CNS to be a meaningful concept - so that an increase in 'cortical inhibition' is taken to have the same effect whether it is produced by depressant drugs, brain damage, or presumably sleep - is no better at explaining the pattern of HRR habituation. Clearly such a crude model is of no use in explaining the results we have here for the HRR.

Lacey (1967) has pointed out, however, that the cardiovascular system has special feedback mechanisms which make the response of the system to stress different from that of the electrodermal system. The properties described by Lacey will not explain the habituation patterns of the HRR, but they do suggest that perhaps it is the peculiarities of the cardiovascular system which cause the HRR not to habituate in REM sleep or drowsiness - rather, that is, than some special property of these states as regards habituation in general. The SPR, after all, shows habituation in all states.

Various ways in which the findings for the HRR in REM sleep might be the result of artifact were considered earlier. The artifacts considered were possible physiological reasons why habituation might not be detected in REM sleep. It now remains to consider various explanations common to this study and to that of McDonald et al (1964) as to why no HRR habituation was observed both in drowsiness and in REM sleep.

McDonald et al ruled out the operation of the 'Law of initial values' (LIV)/

/(LIV) because there were no differences in resting heart rates between their groups. In this experiment, prestimulus heart rates were highest in drowsiness and in stage 4, and lowest in REM and stage 2 sleep. Thus there is nothing in common between drowsiness and REM sleep by way of prestimulus heart rates which could explain the failure of HRR habituation in these states.

McDonald et al also considered the possibility that the HRR was in fact merely a measure of sinus arrhythmia and that differences in the latter between states might possibly have explained the differences in their results between alert and drowsy states. They disproved this by reference to the 'responses' they measured following 'dummy' stimuli. The values for such 'pseudoresponses' in this study are given in Table 18. When these are compared with the response magnitudes on trial one it seems possible that in this study the responses measured in drowsiness and REM sleep were merely a product of sinus arrhythmia. However, the very form of the HRR in drowsiness and REM sleep suggests that these were real responses. Had the 'responses' scored been merely random variations or sinus arrhythmia, the HRR measured would not have had the defined form it presents in Fig. 5.

Could the lack of HRR habituation in drowsiness and REM sleep have been due to the response in these states being a 'startle' reaction (which might not habituate) rather than an orienting reaction? Graham and Clifton (1966) and Graham and Jackson (1970) have discussed the distinctions between startle reactions, ORs, and/

/and defensive reactions. Dykman et al (1959) reported that drowsy subjects were more easily 'startled' than alert subjects. However, like McDonald et al we can rule out this 'explanation' for the findings because if a non-habituating startle reaction were occurring in the daytime control group (or in REM sleep) then the SPR would not have habituated. Moreover, like McDonald et al, trials showing evidence of muscle artifact were excluded from the data analysis in these experiments.

A more reasonable explanation of the failure of the HRR to habituate, under conditions when the SPR did habituate, is that the HRR in these conditions might have been an 'adaptation reaction' rather than an 'orienting reaction'; an 'adaptation' reaction would not be expected to habituate. Meyers and Gullickson (1967) discuss the formation of adaptation reactions in their study of the waking HRR. Sokolov (1963a) has also discussed adaptation reactions, but he does not discuss in any detail the possible form of the HRR in an adaptation reaction. From Lacey's hypothesis (that stimulus rejection is associated with heart acceleration) one would predict that a heart rate adaptation reaction would take the form of an acceleratory response. No skin potential adaptation reaction is to be expected to a pure tone, however. Following Meyers and Gullickson's suggestion one might suppose that in the drowsy subjects, and in REM sleep, an initial OR might be replaced by an adaptation reaction, this masking habituation of the heart rate component of the OR while not affecting SPR habituation. If this were the case, one would predict that the form of the HRR after 20 trials would include an acceleratory component in/

/in drowsiness and REM sleep which would not be present in the NREM sleep states. Inspection of Fig. 5 does not enable us to determine this point with confidence, but it would appear possible that in the daytime controls the initial response is being replaced by the acceleratory response that Meyers and Gullickson termed an adaptation reaction. However, it does not appear that this response is occurring in REM sleep, where a diphasic response remains after twenty trials. We are therefore no further forward in understanding the behaviour of the HRR in REM sleep. (Not that the problem would have been solved if one could tie down the HRR in REM sleep to being an 'adaptation' reaction; we should still have to explain why adaptation reactions occurred in waking, drowsy and REM states but not in NREM sleep).

2. Failure of habituation in other 'states':

It is interesting to note reports of failure of habituation in 'altered states of consciousness'. Kasamatsu and Hirai (1966), in a carefully controlled study, showed that during zen meditation ('zazen'), the brief EEG alpha blocking response to stimuli does not habituate. Wallace (1970a) says that habituation of the alpha blocking response does not occur in transcendental meditation either (although he offers no supporting evidence). Anand (1961) reported that in yogis practising meditation the alpha blocking response failed to occur while they were actually meditating; at other times in these subjects the alpha blocking response occurred, but did not habituate.

Johnson (1970, p.504) in a psychophysiological review refers/

/refers to zen and yogi meditation as distinct 'states' comparable with waking, drowsiness and sleep states. Wallace (1970a,b) has proposed that transcendental meditation be considered a 'fourth state' along with wakefulness, REM and NREM sleep. His argument appears superficially plausible - for instance, basal skin resistance was ^{raised} ~~raised~~ by up to 500% upon resting waking levels in some subjects, whereas rises during sleep are typically only about 100% relative to waking levels (Tart, 1967). However, close inspection of his data on such measures as skin resistance, oxygen consumption, and EEG, suggests that the transcendental 'state' may not be essentially different from NREM sleep stage 1 - or a state of drowsiness. Skin resistance during Wallace's "eyes closed" control condition nearly reached levels while meditating in some subjects. Unfortunately, although Wallace included an "eyes-closed" control condition with his subjects, as well as a meditation condition, he did not use any non-mediating control subjects. Use of such a control would have lent more weight to his conclusions.

Kasamatsu and Hirai (1966) did use such control subjects. They showed that the EEG of zen masters during meditation is strikingly different from the waking EEG. In particular, long trains of theta activity (of 60-70 microvolts) develop. This might seem to resemble a stage 1 drowsy state, as bursts (though not long trains) of theta can occur in stage 1 with an amplitude of over 100 microvolts, However, the two states can apparently be discriminated by zen disciples, for the drowsy state (known as 'konchin') is suppressed during zen training.

If meditational states and states of drowsiness can in fact be thought of as physiologically similar (in the sense in which sleep stages 2, 3 and 4 can be seen as comprising one 'state' NREM sleep), then the failure of EEG reactions to habituate in meditational states is in line with the failure of cardiovascular responses to habituate in drowsiness, found both by McDonald and co-workers (1964) and by Sokolov and co-workers (1961 , 1963).

3. Differences between autonomic and EEG responses:

A second point arising from the results is noteworthy and somewhat curious; there is apparently no effect of ISI on autonomic response habituation, but a fairly clearcut effect on EEG response habituation. It remains possible that an effect of ISI could have been demonstrated if longer ISI conditions had been used (say 45, 60 or 90 sec): one cannot prove a negative. However, the effect was evident with EEG responses even between a 10 sec ISI and a 20 second interval.

Incidentally, the findings here for EEG K-complexes, namely rapid habituation with 10 sec ISIs and almost none with long intervals (30 sec), are in substantial agreement with Pampiglione (1952), who reported habituation only with ISIs of less than 10 seconds. The results here are also consistent with Tizard (1968) and Johnson and Lubin (1967) failure to detect habituation of K-complexes, since they used irregular ISIs of 30 sec or more.

Normally, both EEG responses and autonomic responses are considered components of "the orienting reaction". However, it/

/it can be misleading to assume that various similar responses are in any way part of a single response pattern. "In view of the difficult behaviour of the several variables, it is clear that no one of them could be taken as an index of the activity of the autonomic system, or even of one of its major divisions" (Davis, Buchwald and Frankmann, 1955). Lacey (1967, p21ff) in particular has stressed that it is dangerous to assume that autonomic responses are part of a unitary system (the unitary system Lacey refers to is that of "arousal"). Lacey stressed the dissociation between autonomic responses, where correlations between different autonomic measures are often notoriously low.

Moreover, Furedy (1968, 1969) has shown that for instance in alert adults plethysmographic responses may not habituate under conditions when the GSR will. Furedy is led to the conclusion that "until the behaviour of the electrodermal and plethysmographic components of the OR ... is better understood, it is premature to subsume too wide a class of autonomic responses under the rubric of the orienting reflex".

Why do components of a supposedly unitary OR dissociate at all? I am inclined to conclude that there is indeed no "orienting reaction", merely various responses which may occur to novel stimuli which are not of undue intensity. It is noteworthy that despite Lacey's remarks, curiously little attention has been paid to whether it is meaningful to talk of an OR. Despite several studies in the West recently which refer to the different behaviour of different responses, many/

/many authors continue to refer without question to the 'EDR-OR' or the 'EEG-OR'. The unitary concept of arousal has been challenged; it is time the unitary concept of the orienting reaction was challenged.

Seeing EEG responses and autonomic responses as separate does not, though, help us to see why EEG responses but not autonomic responses should be so sensitive to the effects of ISI. Stern (1968) discussing the lack of relationship between different measures of habituation, was unable to progress further than "the notion of separate response systems under varying degrees of cortical control".

It is difficult even to tie up the results on the effect of ISI here with those from waking, since most EEG studies in waking relate to evoked potential studies, where ISIs are fractions of those used here. Orr and Stern (1970) have, however, directly compared EEG alpha blocking with electrodermal response habituation in a study involving the effect of ISI (regular or random). They too found that EEG response habituation was affected by the ISI condition while the electrodermal response was not. They too were able to offer no more by way of explanation of their findings than Stern's statements quoted above.

This lack of available theory may be related to the fact that there is no general agreement about the effect of ISI on autonomic responses in waking, anyway. Some maintain that response magnitudes are solely a function of the number of trials presented, habituation being 'faster' with short ISIs only in the sense that later trials are reached sooner than with long ISIs. Thompson and Spencer/

/Spencer (1966) and Lader and Wing (1966) hold to this view. On the other hand, Coombs (1938), Martin (1960), Winokur et al (1962), Schaub (1965), Geer (1966), and Grings and Schell (1969) for instance all offer evidence that in fact shorter ISIs lead to faster habituation even when expressed in terms of the number of trials to habituation. Therefore it does indeed seem strange that ISI did not appear to affect habituation of autonomic variables in this study. The fact that ISI effects were clearly evident for the EEG variables suggests that this result was 'genuine', not merely due to a lack of sensitivity of the experiment. However, with no literature (except for the report of Orr and Stern, 1970) making any systematic comparisons of the effects of ISI on both EEG and autonomic responses, there is little to guide us in seeking an explanation of this strange result.

CHAPTER TEN

CONCLUSION

These experiments have demonstrated that habituation of both autonomic and EEG responses is indeed possible in ongoing sleep, at least in some normal subjects: habituation and sleep are not incompatible. But while skin potential responses to the tones habituated reliably in all states, the evoked heart rate responses did not habituate in the daytime controls, when subjects were drowsy. In REM sleep there was some evidence of heart rate response habituation on early trials, but responses became irregular on later trials and over all trials habituation was not significant. Habituation was striking, by contrast, in NREM sleep stages. It would appear that in drowsiness an adaptation reaction (Lacey's "stimulus rejection") in the form of heart rate acceleration may have developed. This may have masked habituation in drowsiness; in REM sleep, however, such a reaction does not seem to have developed: heart rate responses merely became irregular.

In finding EEG response habituation these results are in agreement with most previous studies. With EEG responses the effect of inter-stimulus interval was critical, habituation being markedly retarded when stimulus intervals were as long as 30 seconds. This fact would certainly explain why Johnson and Lubin (1967) and Tizard (1968) were unable to detect habituation of EEG responses.

It is argued that the reason these results do not agree with those of Johnson and Lubin as far as autonomic responses are/

/are concerned, is because their methods of data analysis are seriously flawed. The reason these results differ from Tizard's failure to find habituation of skin potential responses is unclear. Her use of long irregular stimulus intervals cannot be the explanation, for no interstimulus interval effect was found here for autonomic responses. However, since Tizard herself did find habituation in an earlier study with adults (1966), it may be that the age of her subjects (8-10 years in the 1968 study) may be the critical variable - though there is no reason to expect habituation to be more difficult in children's sleep than adults'.

The only difference between these results and those of the one other study, besides Johnson and Lubin's, to look at each sleep stage separately is that while McDonald and Carpenter (1966) found heart rate response habituation in all stages of sleep, habituation in REM sleep was not found here. This difference may lie only in our methods of analysis however, since in this study there was evidence of an initial decrease in response in the first few trials.

The different behaviour of the cardiovascular, electrodermal and EEG response measures was striking. Considerations of these differences between what are often considered 'components' of the orienting reaction leads to the conclusion that it is only confusing to speak of "the OR" - the evidence for a unitary concept of the OR is really very slight, and has not been sufficiently called into question.

B. THE EFFECT OF DRUGS ON DREAMS



CHAPTER ELEVEN

THE EFFECTS OF DRUGS - ESPECIALLY HYPNOTICS - ON REM SLEEP

1. Stimulants, depressants and others

Most psychoactive drugs affect sleep. Most of those which do so affect REM sleep. When REM sleep was thought of as essentially 'light' sleep, it was supposed that stimulants might enhance stage 1-REM sleep at the expense of stages 3 and 4, and depressant drugs do the reverse. Gresham, Webb and Williams (1963) tested this idea on the grounds that stimulants led to enhancement of high frequencies in the EEG, and depressants the reverse. They used alcohol and caffeine, and found that though alcohol significantly reduced the amount of REM sleep, caffeine had no effect. In 1964 Rechtschaffen and Maron showed that amphetamine, another stimulant, actually decreased the proportion of the night spent in REM sleep. Moreover, by also examining the effect of 100mg pentobarbitone with and without 15mg amphetamine, they managed to show that the reduction of REM time by amphetamine was not just due to its action in disturbing sleep - the proportion of REM sleep as well as its absolute amount is reduced if total sleep time falls below about 290 minutes in normal records (Lewis, 1969a).

Baekeland (1967) has since confirmed Rechtschaffen and Maron's findings on the effect both of amphetamine and pentobarbitone. Small, Hibi and Feinberg (1971) recently found that up to 20mg amphetamine in hyperactive children increased the delay to REM sleep, although changes in the amount of REM sleep in the whole night were not significant. Derivatives of amphetamine have also/

/also been shown to reduce REM sleep, in particular diethylpropion (Tenuate), tranlycypromine (Parnate), methylphenidate (Ritalin). (Oswald, Jones and Mannerheim, 1968; Le Gassicke et al 1965; Oswald, 1968 and Baekeland, 1966). Fenfluramine by contrast is one amphetamine derivative that does not seem to reduce REM sleep in small doses (Oswald, Jones and Mannerheim, 1968; Lewis 1969a), but 80mg/day does reduce REM sleep, in common with other amphetamine derivatives.

When these drugs are withdrawn, REM sleep "rebounds" to above normal amounts - whether or not complete tolerance to the REM suppressing effect has developed (Oswald and Thacore, 1963; Oswald, 1968). Nightmares may develop at this point (Le Gassicke et al 1965).

Not only alcohol, but most other hypnotics appear to reduce REM sleep, and delay its onset at the start of the night. In a study by Yules et al (1966), REM sleep was reduced on the first night of alcohol administration, but REM sleep amounts rose to above normal values on further nights of alcohol. Withdrawal also entailed large amounts of REM: it appears possible that a withdrawal effect developed during continued administration. It is particularly possible that an 'intra-night rebound' could develop given a drug which is rapidly metabolised and to which some tolerance has already developed (Kales et al 1971). In clinical studies, Greenberg and Pearlman (1967) reported suppression of REM sleep by alcohol, and increases of up to 100% REM sleep on withdrawal just before the onset of delirium tremens. (This occurred with sleep-onset REM directly following stage 1 without intervening stage 2 sleep). Gross et al/

/Gross et al (1966) reported from 5 years clinical experience that nightmares developed in some patients still on heavy doses of alcohol. One presumes that this might be related to the findings of Yules et al (1966) just described.

It is not only the hypnotic drugs and the amphetamines which reduce REM sleep. Imipramine and its derivatives all reduce REM sleep, and the phenothiazines will also reduce REM sleep at least in certain doses (Lewis and Evans, 1969; Oswald, 1968). Morphine and its derivatives also strongly suppress REM sleep (Lewis et al 1970) as do mono-amine oxidase inhibitors (MAOIs); phenelzine in fact totally blocks REM sleep if given in doses exceeding about 45mg for a period of two or more weeks (Akindele et al 1970).

There are very few drugs which lead to an increase in REM sleep. Reserpine will (Hartmann, 1966; Coulter et al 1971), but there is suggestive evidence that the increase is in fact a "withdrawal rebound" type of phenomenon, resulting from the extreme rapidity with which reserpine is metabolised (Oswald, 1969c). Tryptophan also increases REM time (Hartmann, 1967, 1971; Oswald et al 1966; Evans and Oswald, 1966). It may be speculated that tryptophan is unusual in producing a direct increase in REM time because it is able to directly and immediately increase the rate of 5-hydroxytryptamine synthesis (Oswald, 1969e).

LSD (lysergic acid diethylamide) has been shown to potentiate REM sleep, at least in the first half of the night (Green, 1965; Muzio et al 1966). Muzio et al found that it led to abnormally long first and second REMPs when given just before sleep - though/

/although REM sleep was depressed in the latter half of the night. Durations of the second REMP were in particular excessive, lasting over two hours (Dement and Kleitman (1957a) report upper normal limits for the second REMP of 50 minutes). LSD also led to an increased tendency to awakenings.

The mode of action of LSD on REM sleep is not known. It is a drug which naturally attracts interest on account of its dramatic psychological effects. Similar interest has recently attached to cannabis. Any presumption that its effects on sleep might have been similar to those of LSD has been unfounded. Such a presumption rests upon the idea that both substances are "hallucinogenic"; this term might legitimately be used to describe LSD and mescaline, but is inaccurate in reference to cannabis - which might be termed "illusionogenic", but is probably best considered an "intoxicant" (Ministry of National Health and Welfare, 1970). Cannabis and THC (tetrahydrocannabinol) have been shown to produce either variable effects on different subjects, with a tendency for reduced REM sleep (Pivik et al 1969), a moderate reduction in REM sleep (Bobon et al 1972; Kales et al 1972), or a drastic reduction in REM sleep when given orally in moderately large dosage to two subjects (unpublished observations in this laboratory). Kales et al gave marijuana (smoked) on two periods of three nights each to eight subjects, and noted reduced REM sleep and increased delay to the first REMP on the first night, with tolerance developing by the third night, and an increase in REM sleep with reduced REM latency on withdrawal. Withdrawal/

/withdrawal of marijuana in chronic users also led to an increase in REM sleep. This appears to have been a well controlled study with at least an attempt to provide a smoking placebo. Half the eight subjects were naive and half experienced users.

2. Barbiturates:

When barbiturates are administered, suppression of REM sleep also occurs on the first night on drug. Oswald, et al (1963), Baekeland (1967), Hartmann (1968), Lehmann and Ban (1968), Haider and Oswald (1971), Allnutt and O'Connor (1971) and Kay et al (1972) all used single night studies or balanced designs of one sort or another in their studies of barbiturates. The barbiturates used varied - 400mg heptabarbitalone (Medomin), 100mg pentobarbitalone (Nembutal), 100mg quinalbarbitalone (Seconal), and 200 and 400mg amylobarbitalone (Amytal), but the effects were consistent: reduction of absolute REM time, reduction of the percent of total sleep spent in REMs, increases in the time between sleep onset and the onset of the first REM. The only study not reporting these findings was that of Allnutt and O'Connor; they did report a decrease in REM time, but did not find it significant. However, it seems that their controls were unusual ones. Although the subjects were given two 'baseline' nights initially which were not used in the comparisons, the percent of REM sleep on the 'control' nights (no drug, and placebo) was only just over 15% - well below the normal range of 20-25%. The reason for this presumably was that since the study was related to the performance of pilots on shift duty, subjects were allowed to sleep from 2000 hrs to 0300 hrs only, and they only got just/

/just over 5 hours sleep on the non-drug nights. (These remarks are not made critically however - the experiment was not intended to be an experimental trial, but a test of the efficacy of the drug in a real-life situation).

Administration of barbiturates for more than one night leads to the development of a certain degree of tolerance to the REM suppressant action. As mentioned earlier, with repeated administration of alcohol, REM sleep not only returns to, but actually rises above baseline values. This latter phenomenon does not occur with barbiturate administration. Oswald and Priest (1965) gave 400mg amylobarbitone for nine nights, followed by 600mg for a further nine nights. REM sleep was markedly reduced by the initial dose, but tolerance developed to a large degree (as does tolerance to the hypnotic effect), so that by the ninth night REM sleep was nearly back to baseline values. When the dose was increased to 600mg this pattern was repeated. Withdrawal produced high REM times (up to 40% of the total night's sleep), short delays to REM (less than 45 minutes) and nightmares. Evans et al. (1968) confirmed these findings for 200mg amylobarbitone, showing that over 26 nights on a constant dose REM sleep returned to normal within a week and remained at normal values (note the contrast with the effect reported for alcohol).

Evans et al. also looked at the sleep of a subject dependent on 600mg of Tuinal (equal mixtures of amylobarbitone and quinalbarbitone). He had been taking Tuinal for three years, and had been on 600mg for several months. His REM sleep was unusually low in proportion to a normal night's sleep, indicating that complete tolerance does not develop to barbiturates at these doses. Withdrawal led to a doubling of time spent in REM sleep (besides acute insomnia). Kales et al. (1968b) looked at a subject who had been on 1000mg pentobarbitone for ten/

/ten years. He was withdrawn slowly, but even so he soon obtained up to 40% of his sleep as REM after having had only 7-10% while on the drug; also, whereas he had reported dreaming very rarely on the drug, he reported several nightmares on withdrawal.

Kales et al (1971) studied pentobarbitone 100mg for three successive nights (methyprylon and glutethimide were also included in the study). They found a suppression of REM sleep on the first night, with an apparent return to normal on the second and third nights. On inspection, however, they found a reduction in REM sleep in the first two thirds of the night with a 'rebound' in the final third of the night on these second and third nights.

3. Non-barbiturate hypnotics:

Kales et al (1969a) and Oswald (1969a) have both reviewed their own (and others') studies of both barbiturate and non-barbiturate hypnotics from a clinical perspective. Oswald (1968) has offered a more general review of the effects of drugs on sleep which includes a discussion of non-barbiturate hypnotics. Chloral hydrate was one hypnotic reported not to influence REM sleep at the time of Oswald's review (1968), but it has since been shown to reduce REM sleep (Evans and Ogunremi, 1970). They also tested Mandrax (methaqualone and diphenhydramine) and found no consistent effect on REM sleep. Goldstein et al (1970) also reported that methaqualone had no significant effect on REM sleep during drug administration. However, they did report a significant increase in REM sleep on withdrawal, and Haider and Oswald (1970) reported sleep-onset REM and high REM times after a Mandrax overdose. Flurazepam in a small dose of 30mg/

/30mg appears the only non-barbiturate hypnotic which does not affect REM sleep (Kales et al 1971), of the chemically manufactured compounds. (Either hot milk or the cereal beverage 'Horlicks' would appear capable of acting to maintain sleep without any side-effects on REM sleep - Brezinová and Oswald, 1972). There has been some discussion of tryptophan as a natural hypnotic (Hartmann, 1971), but as mentioned earlier, it appears to be one of the very few drugs to increase REM time, at least at the start of the night (e.g. Oswald et al 1966; Evans and Oswald, 1966; Hartmann, 1967, Hartmann et al 1971).

Nitrazepam is one of the non-barbiturate hypnotics which have been alleged not to affect REM sleep (Hartmann, 1968b). This suggestion derives from a study by Tissot (1965) in which 30-40 mg nitrazepam (Mogadon), diazepam 10-30mg (Valium), - both benzodiazepine derivatives - a "barbiturate" (unspecified) of 250-500mg strength, and reserpine were given to "patients". No information as to the design was given, except that it included 20 control nights, 3 on nitrazepam, 3 on barbiturate, 5 on reserpine and 9 on diazepam. REM sleep was reduced by the barbiturate relative to the control mean of 22%, and increased by all the other drugs. However, only for reserpine and diazepam was the change significant, apparently. The fact that a mean of only 3.0 REMPs occurred on control nights (normative values for eight hours undisturbed sleep would be 5 REMPs), compared with an average of over 4 on nitrazepam suggests that maybe total sleep time varied so much between groups as to make meaningful comparison impossible. Certainly in the absence of any description/

/description of the experimental design, or any data other than the % REM sleep and number of REMPs, it is difficult to lend much credence to the results.

By contrast, Oswald and Priest published in the same year (1965) a lucid study of the effects of 15mg nitrazepam (more nearly anclinical dose than the large dose of 40mg used by Tissot), in a study lasting 14 nights with two subjects. REM sleep was reduced to one quarter of baseline values on the first drug night, and recovered steadily over the fortnight to just less than pre-drug values. The pattern of withdrawal followed that already described for the barbiturates, except that the maximum of the REM "rebound" did not occur until the third withdrawal night instead of on the first following the barbiturate. The presence of drug-induced activity in the EEG persisting until the third night on withdrawal suggested that nitrazepam was eliminated more slowly from the brain. I would speculate that the lack of nightmares following nitrazepam and a maximum REM 'rebound' of only 35%, as against 40% of TST following barbiturate, might be related to the slower elimination of the drug.

Lob et al (1966) reported an increased delay to REM sleep following 10mg of nitrazepam in a two night study comparing the drug with no medication. Unfortunately their drug effect was totally confounded with adaptation to the laboratory, since they only had their subjects in two nights, on the first of which they received nothing, and on the second of which they received the active drug. Total sleep times were so low on the first night (just over 4 hours) and not much better on the next (just over 5 hours) that the value of the study is somewhat dubious.

Lehmann and Ban's study (1968) has already been mentioned. In this carefully designed study, using 4 active drugs and placebo administered in a latin square design, with an adaptation night and each recording night separated by one week, they reported that REM sleep was reduced by 10mg nitrazepam to 15% of total sleep as against 23% on placebo. The onset of the first REM was significantly delayed, but REMs followed each other more rapidly in the latter half of the night than they did on placebo. Haider (1969) and Haider and Oswald (1971) describe essentially similar results using the same type of design - i.e. single night administration spaced a week apart in a balanced design. Allnutt and O'Connor's study (1971) has already been discussed in connection with the effects of barbiturates. Although they found no effects on REM sleep, only a hypnotic effect, their conditions of sleep were unusual. However, this study was double-blind, which many of the previous studies were not. None of these single night studies, however, provide as much information as the extended trials of the sort used by Oswald and Priest (1965), where not only can the effects of a single dose be studied, but also the effects of tolerance and withdrawal effects. Such experiments clearly give information which is of more value both clinically and theoretically than single-night studies. It is my view that the disadvantages of "long-term" studies - in particular the amount of work and consequent possibility of using only a few subjects - are outweighed by the greater value of the information obtained.

One other method of examining the effects of drugs on sleep is to study the effects of acute overdosage. Haider and Oswald (1970) examined seven patients who had taken various overdoses: Mandrax (methaqualone and diphenhydramine), Noludar (methypylon), Mogadon (nitrazepam), phenobarbitone, and aspirin (acetylsalicylic acid). All of the hypnotics produced sleep with abnormally high proportions of REM sleep within two to three weeks after the overdose; aspirin gave rise to only a slight increase, which cannot necessarily be attributed to the drug, as it occurred just before the onset of menstruation, which is accompanied by a slight increase in REM sleep (Hartmann, 1966). All the post-overdose REM sleep "rebounds" can be seen as withdrawal phenomena occurring as the drugs were eliminated from the brain (judged by the presence of drug induced fast activity in the EEG), and consequent upon the development of a certain level of tolerance to the drugs. Although these were large single doses (not all patients had been using the drugs regularly), it is reasonable to suppose the development of tolerance to a drug whose effect is still present up to two weeks after the overdose: phenobarbitone was present in the blood for at least 18 days post-overdose.

4. In general:

All the hypnotics used in the overdoses described by Haider and Oswald (1970) had the same effect - a suppression of REM sleep initially (including periods of coma) followed by a rebound increase in REM sleep at the time of 'withdrawal' of the drug. In almost all drug studies where REM sleep is affected, the rebound on/

/on withdrawal exceeds the amount of REM sleep 'lost' on the drug. This is in contrast to the 'compensatory' rebound which follows behavioural REM sleep deprivation, where the REM sleep 'lost' is never quite made up (Oswald, 1969c).

There are two categories where it would appear that the REMA rebound on drug withdrawal does not exceed that 'lost' on the drug. If considerable doses of barbiturates are taken over a long period (for instance the cases studied by Kales et al 1968b - 1000mg for 10 years - and Evans et al 1968 - 600mg for three years-) then REM sleep remains below normal while the drug is taken, and this "deficit" is apparently never made up following withdrawal.

Secondly, in studies of the effects of mono-amine oxidase inhibitors (MAOIs) on sleep (Wyatt et al 1969; Akindele et al 1970), it has been found that total suppression of REM sleep for weeks at a time is followed by only small rebounds. What is more striking about the rebounds following REM suppression by phenelzine is the short duration of the increase in REM sleep. This is especially noticable in the results following administration of 60mg daily by Akindele et al. The total duration of abnormally high REM sleep barely lasts 10 days (though one subject did have early REM onsets for 20 days). This stands in contrast to a normal time course of up to six weeks following drug withdrawal (Oswald, 1969c). While rapid excretion of drug from brain would appear to be associated with an early peak of REM rebound (Haider and Oswald, 1970), the time for REM sleep to return to normal is usually independent of the circumstances of withdrawal. I am tempted to conclude that/

/that the lack of a typical REM sleep response following phenelzine (45mg or more) is related to the absence of any tolerance to the drug on the part of REM sleep mechanisms. That the development of withdrawal symptoms is intimately related to the previous development of tolerance is widely accepted.

5. The effects of drugs on the profusion of eye movements in REM sleep:

Oswald et al (1963) showed that not only did barbiturates reduce the proportion of the night spent in REM sleep, but they also reduced the profusion of eye movements (EMs), within those REMs. The number of eye movements was counted by one of those authors in ignorance of the drug status of the record. Of the subjects who received 400mg heptabarbitalone, the mean number of eye movements in the second and third REMs was reduced to some 55% of the number on placebo. A slight increase was recorded in the subject who only received 200mg.

Baekeland (1967) confirmed this finding when he compared the number of 2 second epochs in the first two REMs of the night on placebo and after 100mg pentobarbitalone. The percent of 2 sec epochs with EMs was reduced from 7% and 9% (in REMs 1 and 2 respectively) on placebo to 1.3% and 2.2% on pentobarbitalone. Both changes were significant. Lester et al (1968) and Feinberg et al (1969) have also confirmed that barbiturates reduce eye movement profusion. Amphetamine, although its effect when combined with barbiturate on the percent REM sleep is highly significant (a reduction from 14% to 4.5%), has no further effect upon the profusion of eye movements (Baekeland, 1967).

Small, Hibi and Feinberg (1971) examined the effect of amphetamine per se on the profusion of eye movements in hyperactive children. Although there were no significant changes in the proportion of REM sleep in the night in these children (i.e. they differed from normal adults, in whom amphetamine reduces REM sleep), the percentage of 4 sec epochs with EMs was slightly but significantly increased by 20mg amphetamine. Values after 5mg, and after withdrawal from 20mg, were not different from baseline (40 % epochs with EMs); 20mg increased this to 48%. Since in this study the drug was given by day, it is possible that the increased profusion ~~during~~ during the following night was a "withdrawal" effect. This would be a little strange, however, since in Baekeland's study night-time administration of amphetamine did not reduce EM profusion.

Reserpine also has effects on EM profusion which have no ready explanation. Coulter et al (1971) found that although reserpine increased REM sleep in repeated doses, it significantly decreased the number of 10 second epochs in REM sleep with eye movements both in single doses (when the effect on REM sleep was not significant) and in repeated doses. These effects of amphetamine and reserpine are unusual. Other drugs appear to affect the profusion of eye movements within REM in the same sense as they affect REM sleep itself. Thus Lewis (1968b) found that nitrazepam 15mg, although only reducing the number of EMs on the drug by some 10% within a five minute period, had a dramatic effect on withdrawal. While the time spent in REM sleep is less than doubled on withdrawal from this dose, the number of EMs was increased to more than five times the number in the same/

/same period of time in the control condition. Maxion and Schneider (1971) also reported that the number of EMs per minute was doubled in patients up to 8 days after acute alcoholic psychoses, when REM sleep is generally more prevalent. Evans and Oswald (1965) also reported that the profusion of eye movements was increased following the administration of tryptophan to narcoleptics. Not only were their initial REMPs doubled in length, but the records could be assigned to control or tryptophan conditions by inspection of the EOG by a judge unaware of their status. Kales et al (1969a) also report that the profusion of EMs is disproportionately decreased by glutethimide (which reduces REM sleep percentage), and disproportionately increased on withdrawal. Goldstein et al (1970) also report a significant decrease in the number of EMs on glutethimide.

CHAPTER TWELVE

RELATIONS BETWEEN THE DREAM AND THE PHYSIOLOGY OF THE REM PERIOD

1. Eye movements and dream imagery: the "scanning hypothesis":

"But I am inclined also to believe that, in somewhat vivid visual dreams, the eyeballs move gently in their sockets, taking various positions induced by the retinal phantasms as they control the dreams. As we look down the street of a strange city, for example, in a dream we probably focus our eyes somewhat as we should do in making the same observation when awake, though with a complete lack of that determined teleological fixedness which in waking life comes with it".

G.T. Ladd (1892), contribution to the psychology of visual dreams.

Berger (1969) has suggested that there may be no difference between the nature of dream experiences in REM and NREM sleep, and that the only reason REM sleep has become associated with dreaming is because of differences in our ability to remember what went on during REM and NREM sleep. This viewpoint lies at the extreme end of those who regard a specific connection between REM sleep and dreaming as tenuous at least. The assumption here is that we know little or nothing about what 'really happens' during either REM or NREM sleep - what is important is why we forget (or remember) any of it. In this frame of reference, Webb and Kersey (1967) set out to show that the probability of recalling a dream in the morning was primarily dependant upon the probability of waking from a REM - on the/

/assumption that NREM dreams do not contribute to daytime recall. The emphasis here is on the relation between dreaming or dream recall and REM sleep as a tonic state, with less emphasis upon what goes on within the REMP.

The contrasting approach to dreaming is that which attempts to find out exactly what does 'really happen' during REM sleep. Interest here tends not unnaturally to focus on the most dramatic externally observable events within the REMP: the eye movements (EMs). The first attempt to do this was that of Dement and Kleitman (1957a). After establishing a close correspondence between the subjective duration of dreaming and the duration of the REMP, they further attempted to establish whether there was a specific correspondence between the direction of the EMs in the REMP, and the direction of the dreamer's gaze in the dream. The subjects were awakened as soon as one of four dominant patterns of eye movement had persisted for one minute. These were: mainly vertical EMs, mainly horizontal EMs (these two categories were very rare), a mixture of horizontal and vertical eye movement, or fourthly very little or no eye movement. The one case (in 35) of mainly horizontal movement was associated with a dream in which the subject was watching people throwing tomatoes at each other. The three cases of chiefly vertical eye movement were associated with dreams which involved the subject in looking upwards, or looking repeatedly up and down. These four instances provided the chief evidence upon which Dement and Kleitman concluded that the EMs in REM sleep reflected the changes in the dreamer's gaze, as he looked about his dream world. The/

/The instances with little or no eye movement were associated with dreams in which the dreamer was either watching something at a distance, or staring fixedly at an object - in contrast to dreams where the dreamer was looking at objects close by, which came from REMPs with mixed horizontal and vertical EMs. It should be emphasized that (excepting the four special cases) the distinction between the dream reports from REMPs with eye movement and those without does not establish any connection between the direction of EMs and the direction of gaze. What it does is establish a connection between the presence or absence of EMs and the type of dream content - "close involvement" versus "involvement at a distance". Clearly this has more in common with what has since been termed "active" versus "passive" dreaming than any statements about direction of gaze. It should also be born in mind that no statistical evaluation of the findings was made.

Dement and Wolpert (1958) went on to examine the relationship between body movements and fragmentation of the dream experience - between which they found a highly significant correspondence - and also to re-examine the relation between EMs and dream content. When the dreams and EOG records were separated, coded, and then judged "active" or "passive" independently, a highly significant relation was established between profuse eye movement activity and a particular type of dream content. Further, in 23 instances the hypothesis examined was that EMs should reflect direction of gaze in the dream. Controls against bias in this part of the experiment do not appear to have been noteworthy. The results simply reported were that/

/that "the last eye movement in the record was identical with the last reported fixation in the dream in 17 cases".

Those parts of this experiment which were conducted so as to scrupulously avoid experimenter bias did not, as has often been assumed, concern the relation between dream-gaze and observed EMs. The hypothesis examined was that the presence of ocular activity corresponded with "involvement of the dreamer in the dream". Active dreaming was taken as active participation in the events in the dream; passive dreaming was "quietly reflecting upon an event, talking quietly to another person, or watching an event occur, often from a distance, in which (the dreamer) took no active part". From this description it should be clear that active dreaming refers to immediate involvement in the dream, not to the visual activity of the dreamer. One is struck by the resemblance of this description to that of "secondary cognitive elaboration" used much later by Molinari and Foulkes (1969).

In 1962 Roffwarg, Dement, Muzio and Fisher published a now classic paper in which they set out to test the hypothesis "that there exists a 1:1 correlation between the direction of each eye movement and the direction of each alteration in the hallucinated gaze of the dreamer". Two experimenters were used: one chose to wake the subject when distinctive EM sequences were occurring while the other, who could not see the EOG, performed the interviewing and attempted on the basis of the subject's dream report to predict EMs which would have accompanied it. Three judges then rated the correspondence between the predicted EMs and the real EOG, for/

/for each waking. The correspondence was rated as 'good', 'fair', or 'poor', for each of the 121 usable reports. The proportion of 'good' ratings varied, according to the clarity of the subjects' descriptions from 53% to 80% (he gave each report a "confidence rating"). The proportion of 'poor' correspondences was at most 27%. These results were illustrated with various examples, and a discussion of the reasons why the authors felt that a 100% correspondence was difficult to achieve in practice. These reasons were threefold: failure on the part of the dreamer to recall accurately his shifts of gaze, failure of the dreamer to communicate his dream to the experimenter, and thirdly failure of the experimenter to predict correctly the eye movements which would have been made (in real life, for instance) by the dreamer given his dream report. Considering these factors, Roffwarg et al felt that they had achieved as satisfactory correspondence as could be expected.

Unfortunately, no method of evaluating the results statistically was presented. Had the judges been biased in the hope of obtaining a good correspondence between actual and predicted EMs, they could have produced this result by simply rating all the matchings as 'good'. There was no procedure laid down, and no criteria setting out just how accurate the prediction had to be before it was judged 'good'. There is only one way post-hoc in which one can assess the results. Two of the judges were not given the subject's "confidence level" for his dream recall. The results were presented for each of the three confidence levels for each subject. Now if the judges/

/judges had been systematically rating all matchings "good", the proportion of "good" matches should not differ according to the subject's confidence level. But if a real correspondence existed between dream action and EOG, then one would expect poorer correspondence in those cases where the subject's own memory for events was poor. There was indeed a trend in this direction, and it is even possible to assess this effect. Working from the figures given in their paper, it would appear that judge 1 rated 62 out of 77, 18.5 (?) out of 28, and 8.5 (?) out of 16 matchings "good" in the "clear", "moderate", and "fuzzy" recall categories respectively. Judge 2 rated 58 of the 77, 21 of the 28, and 10 of the 16 matchings "good" in each of the three categories. (Incidentally, the judges agreed on 81% of their ratings). Chi-square calculated from these figures is 4.90 for judge 1, and 1.15 for judge 2. If a one-tailed test is used, judge 1 rates significantly fewer matches as 'good' when the subject's recall is fuzzy than when the subject's recall is clear. (A Chi-square of 4.60 or more with 2 DF has a probability of 0.05 in a one-tailed test). Judge 2 did not differ significantly between recall conditions in the proportion of matchings he rated 'good'. However, it does appear that a better correspondence between predicted and actual eye movements can be obtained when the dreamer's recall is clear than when it is only fuzzy.

A recent 'replication' of this experiment (Jacobs et al, 1970, 1971) apparently failed to confirm Roffwarg et al's results. Jacobs et al woke their subjects after either a single saccade, a burst of saccades, or a period of ocular quiescence. The actual EOG and/

/and the dream reports were then assessed for their correspondence - the details of this procedure are not unfortunately given in Jacobs et al's papers. Correspondence was assessed as "positive", "negative", or "uncertain". Positive correspondences were apparently rare after either ocular quiescence or after a series of saccades. After single saccades, positive correspondences were obtained for 18% of reports, and negative correspondences for 10% of reports (i.e. the eye movements were opposite to those implied by the dream report). In the remaining 72% of cases, there was no relation between eye movements and dream report. Jacobs et al concluded that on the whole EMs in the REM sleep were not related to the action in the dream; it is only to be regretted that they did not provide more information both on their procedures and on their actual results.

An attempt to remedy the problem of bias in the experiment of Roffwarg et al was made by Moskowitz and Berger (1969). In this experiment, the judge was given four dream reports to try to match correctly to four EOG records, which he had selected as representing four different types of EOG activity (predominately horizontal, vertical, or oblique or else alternatively horizontal and vertical). This procedure was repeated with further sets of four to a total of 56 records. The probability of the judge correctly matching records and dreams by chance was exactly one quarter, so a binomial test could be used to assess the probability of obtaining the 18 correct matchings which occurred. 14 correct matches would have been expected by chance, and the probability of obtaining 18 or more was greater than one in eight. The authors concluded that the/

/the EMs of the REMP do not represent the dreamer "scanning" his dream. They noted in particular several instances where the eye movements were completely inappropriate to the dream report. The only criticism which can apparently be levelled against this study is that since the authors were apparently hostile to the idea that EMs in REMPs represent "scanning" of the dream, the judge who attempted to match reports with EOG records could have been simply careless, and thereby prejudiced the chances of getting a significant number of correct matches.

Very recently there has been an attempt (Bussell, Dement and Pivik, 1972) as yet unpublished, to replicate Roffwarg's original study in view of the criticism it has received for 'bias', and in view of the report by Moskowitz and Berger. This study attempted to control for bias in rating correspondence by giving the judge "dummy" pairs of EOG and predicted EMs to rate for correspondence. These sets were generated by scrambling, and by rotating, the predicted EMs in the real pairs. Also, the whole procedure was performed on waking subjects (where we can assume that we really do scan our visual world), to provide another standard for comparison. Apparently the correspondence between EOG and predicted EMs was significantly better for the real than for the "dummy" pairs, and was in fact as good as for the waking condition. There is uncertainty about evaluating this study however until it is published.

It used to be thought that persons with life-long blindness neither had visual dreams, nor eye movements in REM sleep (Berger et al 1962).

However, Gross et al (1965) showed that in subjects who had been blind since childhood, eye movements could not be recorded with a conventional electro-oculogram (recording either DC or AC). The EOG records changes in the corneo-retinal potential field as the eyeball moves in its socket. In those with lifelong blindness however, retinal degeneration develops, and the corneo-retinal potential is therefore no longer observable. However, use of a strain guage attached to the eyelid does enable eye movements to be recorded. It has been argued (Berger, 1967) that the strain guage recordings of Gross et al merely pick up eyelid flutter; however, direct observation of the eyes of the blind in REM sleep will reveal obvious rapid, conjugate movement of the eyeballs beneath the closed lids. Thus it would appear that even in those subjects who have been blind long enough to only have totally non-visual dreams, rapid eye movements do indeed occur during REM sleep. This clearly poses problems for hypotheses in which EMs in REM sleep are supposed to represent scanning movements of the dreamer looking about his world.

2. 'Active' and 'passive' dreams:

The evidence for the "scanning hypothesis" - that EMs in REMs represent the scanning of his visual world by the dreamer - is therefore seen to be rather dubious, with no convincing experiment offering evidence in support yet published.

Another hypothesis relating the EMs of the REM period to the dream content elicited on arousal may be offered. It may be hypothesised that although EMs do not represent the dreamer scanning his visual/

/visual world, there exists a correlation between the physiological 'intensity' of the REMP and the psychological 'intensity' of the dream content.¹ I shall refer to this as the "intensity hypothesis". Specifically, by intensity of the REMP is meant the profusion of eye movements. Various measures of the intensity of the dream are possible. A dream would be regarded as intense to the degree that it includes strong affect, a high degree of active involvement by the dreamer, or a high degree of activity by any of the characters present. In the absence of these indicators, the number of characters, the number of actions or even the number of scenes may be used to discriminate dreams on the concept of 'intensity'. Dreams regarded as lowest in intensity are thus those with few characters who engage in few actions, and those in which active involvement on the part of the dreamer is at a minimum. The intensity hypothesis predicts that all these measures of the 'intensity' of a dream will be positively correlated with the profusion of eye movements in the REMP. What it hypothesises is not a 1:1 relationship between discrete events in the REMP and discrete events in the dream, but some positive correlation between the frequency of 'events' in the dream content and the frequency of EMs in the REMP.

Dement and Wolpert (1958) and Dement and Kleitman (1957a) both offered evidence on this point which has already been mentioned.

¹The concept of a global dimension of 'dream intensity' has been used, for example, by Snyder (1971).

Dement and Kleitman established a connection between lack of EM activity and dreams with "action at a distance" rather than "action close by"; Dement and Wolpert found evidence for a rather similar association of little EM activity with reflection or talking quietly or watching an event at a distance.

Berger and Oswald (1962) also examined the relationship between "active" REMPs and "active" dreams. Berger and Oswald set out to replicate Dement and Wolpert's study of active and passive dreams, and they did indeed find a significant relation between 'active' dreams and 'active' REMPs. However, it is worth noting/

/noting that Berger and Oswald saw themselves at the time as testing the scanning hypothesis - the dream reports were scored active "according to the nature of the events described, and especially if such events would have been accompanied by many shifts of gaze, had they occurred in real life". However, since the more specific tests of the scanning hypothesis, this paper has been regarded rather as supporting the "intensity" hypothesis in the form of Berger and Oswald's conclusion that "there is a significant association between the nature of the dream content and the amount of movement of the eyes". This study was carefully conducted, since the authors felt that in Dement and Wolpert's study those authors could have remembered some of the EOG tracings to which they matched the dream reports: hence in the Berger and Oswald study the judge never saw either EOG records or dream transcripts until he rated them. (However, those who have woken subjects for dream reports will know that one's recall of events during the night is never very good).

Berger and Oswald's study has been criticised from a rather different standpoint. Hauri and Van de Castle (1972) criticised it for confounding the active-passive correlation with the "time of night effect". It is well known that REMPs later in the night have a greater profusion of EMs (e.g. Goodenough et al, 1965b; Aserinsky, 1969). Also, dream content becomes more 'intense' as the night progresses - dreams become more perceptual, vivid, dramatic, emotional and involve more activity on the part of the dreamer when awakenings are made from REMPs later in the night.

The content of dreams tends to refer increasingly to events further away from current or immediate concerns of the dreamer. Situations and characters in dreams late in the night are less familiar from the dreamers experience (Domhoff and Kamiya, 1964a; Verdone, 1965; Foulkes, 1966; Dorus et al, 1971). Thus in various ways the dream experience may be seen to become more 'intense' as the REMPs from which they are drawn are more profuse in eye movements. While this time-of-night effect may itself be taken as evidence for an intensity relation between dream content and EM profusion, in the context of Berger and Oswald's study it is a confounding factor. Controlling for the time of night, is there a relation between 'active' dreams and 'active' REMPs?

Keenan (1970) reported a study purportedly testing the "scanning" hypothesis, in which the relation of the number of 20 sec epochs with EMs to the number of activities was examined. The number of activities was determined from the Hall and Van de Castle system of content analysis (1966). A significant relationship was found from 71 dream reports collected over 15 nights. Only one subject was used. As a test of the scanning hypothesis this seems to have been a curious one, since the Hall-Castle "activities" scale includes activity in which characters other than the dreamer engage - and need bear no relation to the dreamer's visual involvement. As a test of the intensity hypothesis on the other hand it is relevant. Nevertheless, when the results of this experiment were further analysed (now totalling 20 nights, 97 reports) it was found that if the reports from the first REMP of the night were analysed separately,

/separately, the significant relationship vanished (Keenan and Krippner, 1970). This suggests that the relation between active dreams and high EM profusion found by Berger and Oswald (1962) and Dement and Wolpert (1958) may have been an artifact of the "time of night effect".

3. Further relationships between dream content and eye movement activity in the REM period

Besides the supposed association between active dreams and profusion of eye movements, there are other "dimensions" of dreaming which have been linked to characteristics of the REM. Goodenough et al (1965b) looked at "some correlates" of dream reporting. They were particularly interested in correlates of arousal thresholds and eye movement profusion. They found significant correlations between the number of 3 sec epochs containing EMs and an "action rating". Also, the percent of 3 sec epochs in the REM with EMs was on average 5% lower for "rational" than for "bizarre" reports and 5% lower for narratives without "reported visual imagery". Since these correlations were established across the whole night, however, they may merely reflect the time of night effect. Goodenough et al found that 13%, 20%, and 25% of 3 sec epochs had EMs respectively in the first, second, and subsequent REMs. It is also worth noting that these relationships, although significant, represent very slight differences in EM activity. Despite the difference in EM activity for reports with and without visual imagery, there was no difference in EM activity between reports subjects labelled as thoughts or dreams. Goodenough et al conclude on this point "....REM periods which preceded thinking and dreaming reports could,

/could not be distinguished in terms of arousal thresholds or eye movement activity".

In another study examining the determinants of thinking reports by the same group (Shapiro et al. 1965) an examination was made of EM activity preceding gradual awakenings (which are a factor enhancing the probability of thinking reports). Shapiro et al looked to see whether the cessation of EM activity at the start of the gradual awakening stimulus would increase the probability of a thinking rather than a dreaming report. They found a slight trend in this direction, but the effect was not significant.

Pivik and Foulkes (1966) looked at the effect of REM deprivation on dream content. They deprived their subjects of REM sleep in the first half of the night, and then awoke them from REMs in the second half of the night to see what effect the deprivation had, if any, on dreams from subsequent REMs. They also ran control nights with "pseudo-deprivation" - awakenings from NREM instead of REM sleep. Using the Foulkes dreamlike fantasy scale (Foulkes et al 1966) they found an 'intensification' of dreamlike fantasy following early-night REM deprivation, which was significant for their group of high on "repression" although not for their group high on "sensitisation". The effect of REM deprivation on the profusion of eye movements (number of 2.5 sec epochs) was identical - an increase in both groups, which was significant only for the repressors. Pivik and Foulkes concluded by noting that this "close correspondence" between EM profusion and dream "intensity" on the dreamlike fantasy scale tied in with the earlier experiments on active and passive dreams.

However, when Foulkes et al (1968)¹ examined the effect of REM deprivation one night on dreaming the next night, they did not find any significant effect. Indeed, REM deprivation one night had no significant effect on either REM time, or EM profusion the next night, so it was hardly surprising that no effects on dream content were found.

Antrobus et al (1970, 1972)² also examined the effect of REM deprivation one night on dreaming the following night. Their study, however, would not appear to be an improvement on that of Foulkes et al in any way. Antrobus et al preceded their REM deprivation night with four nights of REM suppression (using barbiturate and amphetamine). This procedure was presumably chosen to increase "REM pressure" on the experimental nights (drug withdrawal nights). However, it means that two effects, drug withdrawal and recovery from REM deprivation, were confounded in an experiment supposedly studying the effect of REM deprivation.

In the published report of this experiment (Antrobus et al 1970)² the authors do not in fact discuss dream content at all, but describe only the length of the verbal report. In the unpublished version/

¹Foulkes, D., Pivik, T., Ahrens, J.B. and Swanson, E.M. (1968). Effects of "dream deprivation" on dream content: an attempted cross-night replication. J. Abnorm. Psychol. 73, 403-415.

²Antrobus, J.S., Arkin, A.M. and Toth, M.F. (1970). The effects of REM period deprivation on sleep mentation. Psychophysiology, 7, 332.

Antrobus, J.S., Arkin, A.M. and Toth, M. (1972). The effects of REM deprivation on sleep mentation. Paper presented at meetings of the Association for the psychophysiological study of sleep, New York, 1972.

/version (Antrobus et al 1972)² the authors report that judges ratings of the dream content showed no effect of the REM deprivation, a result which coincides with the earlier findings of Foulkes et al (1968)¹. Where Foulkes et al had measured EM profusion scores, Antrobus et al did not. It is therefore impossible to establish whether the lack of effect on dream content was associated with a lack of effect on eye movement activity.

A paper by Cartwright and Monroe (1968)³ entitled "Relation of dreaming and REM sleep: the effects of REM deprivation under two conditions" is not in fact relevant to the relation between dream content and eye movement activity. Cartwright actually examined the effect of post-awakening activity (reporting previous mental activity or repeating lists of digits) on subsequent recovery of REM sleep. Eye movement profusion was not measured, so no comparisons between dream content and eye movement activity are possible.

Karacan et al (1966) examined the number of 3 sec epochs with EMs in the last 3 minutes of each REM period from which they woke subjects for dream reports. They then divided each subject's REM periods into those above and below the median in EM profusion. They found more total affect in the dreams which came from REM periods above the median in EM profusion when they used the Nowlis checklist to measure affect in the dream. They used six of the seven dimensions in the checklist/

³ Cartwright, R.D. and Monroe, L.J. (1968). Relation of dreaming and REM sleep: the effects of REM deprivation under two conditions. J. Personal. Soc. Psychol. 10, 69-74.

/checklist to score total affect: aggression, anxiety, surgency, social affection, depression and distrust. However, single, only one of these dimensions correlated significantly with EM profusion - aggression. They also found higher EM profusion in those REMPs with full penile erection than in those with either irregular erection or no erection.

Hobson et al (1965) examined the number of eye movements in the 30 sec before awakenings. Rank order correlations with a "total content rating" (emotion, physical activity and vividness) were significant. The correlation was only of the order of .30. It is noteworthy in their figures how little of the variance in the total content ratings was accounted for in relation to the profusion of eye movements.

A recent report by Takeo (1970) based on 640 awakenings from 34 normal subjects attempted to relate physiological indices of sleep to dream content. High profusion of eye movements (3 sec epochs) was associated with high incidence of complexity and colour in dreams; /

/dreams; the dreams also tended to be more distinct (in recall), bizarre and illogical. However, there was relation between the profusion of EMS and whether the reported experience was visual or non-visual.

4. Dream content and other physiological activity:

One striking aspect of physiological activity in the REM sleep with content in the dream is the penile erection. Karacan et al (1966)/

/(1966) showed that dream content high on anxiety came from REMPs with an absence of erection (20% of REMPs), or from REMPs with irregular erection (35%) rather than from REMPs with full erections. Fisher (1966) found that tumescence and detumescence could correctly be predicted from the latent content as well as from the manifest content of the dream.

Attempts to correlate cardiac and respiratory events in the REMP with dream content have been markedly less successful. Fisher et al (1970a) remark on the lack of autonomic activity they observed during the REMPs which gave rise to the only five sexual dreams they recorded in the laboratory (manifest sexual content is much less frequent in the laboratory than at home).

Shapiro et al (1964) attempted to relate cardiac and respiratory activity to dream recall (though they did not look at specific content). They did find that variability of respiration was highest in instances where content was forgotten - which they interpreted as indicating that high respiratory variability was associated with anxiety in the content. However, they concluded that in their attempt to correlate cardiac and respiratory activity with dream recall their findings were on the whole "disappointing". Hobson et al (1965) found no significant correlation between respiratory variability and content, although they did find a small but significant relation with absolute respiration rate, both in REM and in NREM sleep. The specific aspects of content correlated with respiration were the emotions of fear and pleasure. They also reported that specific "respiratory content" in the verbal report was significantly more frequent following apneic respiration. This kind of relation/

/relation has also been found in connection with minor body movements - Wolpert (1960) found some correlation between "amount" of EMG activity and "amount" of physical activity in the dream (he does not say how he scored these "amounts"). However, Wolpert was forced to conclude that "in general the correlation ... if not very impressive ...". Hobson et al were similarly struck by the "non-specificity" of their findings.

Hauri and Van de Castle (1972) have reported highly significant relationships of dream emotionality with heart rate variability and with skin potential fluctuations in REMPs during the last minute before awakening. They also reported some relation between emotionality and both respiratory rate and the number of vasoconstrictions in the minute prior to waking. Their results relating to cardiac variability are opposed to those of Baust and Engel (1970), who reported a negative relation between emotionality and cardiac variability. Hauri and Van de Castle quite convincingly seek to explain this difference in terms of Lacey's individual response specificity - i.e. the fact that different individuals respond differently to stress (Lacey, 1950).

In summary it would appear though that correlations between autonomic activity during the REMP and the ensuing dream are marginal in comparison with correlations between EM profusion and dream content - the former are very much suggestive of the relation between two variables which are only very indirectly correlated through a third. I shall now return to the relations between EMs and dreaming, from a rather different point of view: not that of the correlational/

/correlational approach, but the "microscopic" approach.

5. Microscopic studies of mentation: tonic and phasic events:

Molinari and Foulkes in 1969 lent a new colour to psychophysiological studies of sleep mentation with their paper entitled "Tonic and phasic events during sleep: psychological correlates and implications". (The term mentation becomes useful when one is dealing with verbal reports from both REM and NREM sleep, many of which the subject will not describe as dreams but as thoughts. The one word mentation is useful to describe both of these things.) They started from the fact that PGO (pontogeniculo-occipital) spikes occur both in NREM as well as in REM sleep (Bizzi and Brooks, 1963; Michel et al 1964) and that dream reports can be obtained from NREM as well as from REM sleep (Foulkes, 1962, 1966, 1967). Aserinsky (1965) separated REM sleep into two phases, REM-M and REM-Q, on the grounds that periods of REM sleep with bursts of eye movements (REM-M) showed a different EEG spectrum from that during quiescent periods of REM sleep (REM-Q). Aserinsky (1967) went on to propose that dreaming might be associated with the 'motility' segments of the REM (REM-M), rather than with the quiescent periods of REM sleep.

Taking this prediction as their starting point, Molinari and Foulkes conducted awakenings during active and quiescent periods of the REM, during NREM sleep, and at sleep onset. They stressed that their subjects should concentrate upon the very last experience before they were woken. On the basis of their preconceptions with both REM and NREM mentation, the two/

/two experimenters then devised a system for scoring the reports. They scored the presence of what they termed "secondary cognitive elaboration" (SCE) in the reports. This included active intellectual processes (thinking, being aware), conceptual relationships, alternatives or comparisons, and verbalisation or explanation (the dreamer himself talking or any character in the dream using words by way of explanation rather than description). Reports not containing any of these elements they scored residually as "PVE" (primary visual experience). The term primary visual experience has been misleading, I think, because it suggests comparison with those procedures which have sought to differentiate visual from non-visual mentation. SCE reports too can be very visual; their essence is that they also contain an element of reflection or awareness superimposed. By concentrating on the very last experience Molinari and Foulkes hoped to get the mentation which occurred in conjunction with the burst of EMs - or the quiescent phase - just before the subject was awoken.

One of the two judges was blind as to the awakening categories. Initial agreement on whether reports were SCE or PVE ranged between 93 and 100%. After reconciliation, it was found that only 12% of reports following REM-M wakings were scored as containing SCE. On the other hand, 80% of reports following REM-Q wakings contained SCE. Further, 77% of NREM reports contained SCE, and around 60% of sleep onset reports did so. The incidence of SCE was significantly higher after REM-Q than after REM-M, while there was no significant difference between REM-Q, NREM, or sleep onset reports in the frequency of SCE. These were striking results. As a check, a totally naive judge was given the reports, and agreement between him and the other two then lay between 82 and 90%.

This division of the experience in REM sleep into SCE versus PVE, and their correspondence with the physiological distinction REM-M versus REM-Q, has been confirmed in a replication by Foulkes and Pope (1972), with the proviso that the verbal material to be scored is limited to that produced spontaneously. Thus it is usually possible to find evidence of some cognitive elaboration in material elicited from REM sleep; what is important, however, is that SCE is the salient feature of mentation in quiescent periods of REM sleep, whereas it is not during REM-M.

Further, Foulkes and Pope noted that periods of REM sleep characterised by 3Hz frontal saw-tooth waves (Berger et al 1962) produced mentation like that elicited from REM-M awakenings. Berger et al noticed that saw-tooth waves significantly often preceded bursts of EMs. They remarked then that it seemed difficult to reconcile the presence of these waves, just before bursts of EMs, with the scanning hypothesis. Why should such waves provoke the dreamer into looking about his world?

It cannot escape notice that the occurrence of PVE rather than SCE at the time of occurrence of saw-tooth waves argues strongly against any idea that EMs are uniquely linked to the dreamer's changes of gaze.

A further replication of the SCE-PVE dichotomy from REM-Q and REM-M awakenings has also just been completed in this laboratory by Michael Holmes. In this experiment, which among other things replicated Molinari and Foulkes original paper (1969), two judges (this author, and Mark Austin), both blind as to the awakening/

/awakening conditions, reached 90% agreement after independently scoring 94 REMF reports for SCE versus PVE from the published criteria of Molinari and Foulkes. (Their original report in fact included only 36 REM awakenings). Of REM-M reports, only 11% were found to contain secondary cognitive elaboration.

Foulkes et al (1972) have also tried this technique on childrens' dreams. They found that neither the incidence of visual imagery nor of conceptual activity discriminated the phasic (REM-M) from the tonic (REM-Q) aspects of REMPs. This experiment, in which the authors thus failed to substantiate Molinari and Foulkes' findings, threw up one other curious result. The only variable which did discriminate the phasic from the tonic awakenings was the occurrence of visual activity on the part of the dreamer - a finding which can only be regarded as in direct support of the scanning hypothesis. That these subjects were 10-12 year old children might explain the apparent lack of relevance of the SCE-PVE dichotomy, but it would not seem to be relevant to the finding that looking, reading and watching were significantly associated with bursts of EMs (the criteria used for determining REM-M wakings).

R_chtschaffen and co-workers have recently been exploring the possibilities of using PIPs (periorbital phasic integrated potentials) as indices of phasic activity both within and without REM sleep. Watson (1972) studied the very last experiences before his subjects were woken - during a burst of PIPs and EMs, during a burst of PIPs alone, ~~and~~ during a quiescent period of REMAsleep. Compared with the 'control' quiescent REM reports, reports following PIPs or PIPs/

PIPs and EMs were significantly more often bizarre. Moreover, an increase in bizarreness from the next-to-last experience to the last experience was significantly more frequent following PIPs and EMs, and this was also true of the frequency with which the very last experience did not fit into the context of the previous one. There were no differences between reports following PIPs alone and those following PIPs and EMs. Thus it seems that PIPs occurring during REM sleep are associated with discontinuity in the dream content, and the intrusion of bizarre elements. Rechtschaffen et al (1972) demonstrated essentially the same effects of PIPs in NREM sleep (PIPs can be detected in approximately 50% of 2.5 sec epochs during REM sleep, and anything between 2 and 10% of such epochs during NREM sleep). The noteworthy aspect of these findings is that in REM sleep the presence or absence of eye movements when accompanied by PIPs had no effect upon dream content.

Thus we see that PIPs (but not EMs unless accompanied by PIPs) are associated with the appearance of bizarre elements in ongoing mental activity, and with the occurrence of discontinuities (the last experience not fitting in with preceding events). Molinari and Foulkes have also shown that phasic activity as judged by EM bursts is associated with the cessation of thinking, reflecting, verbalising or explanation in the mental activity. Since most EM bursts are associated with PIPs, these two conclusions can be tied together. Thus we can conclude that phasic activity in the REM sleep is associated with discontinuities in the content of the dream, the replacement of thinking, reflecting and verbalising by primarily visual activity on the part of the dreamer at the same time as bizarre elements appear in the dream.

6. REM and NREM dreaming:

Although this chapter is entitled "Relations between the dream and the physiology of the REM", it is suitable to briefly discuss here the evidence that dreaming is not confined to REM sleep, before the final section on nightmares and REM sleep. Of particular concern here is the nature of any differences in mentation between REM and NREM sleep. I shall not attempt to discuss evidence regarding differences in mentation within NREM sleep, in particular the evidence that mentation in NREM sleep may be affected by phasic events within NREM sleep (Rechtschaffen et al 1972), but wish merely to contrast thinking and dreaming in REM and NREM sleep.

Whereas early workers had contented themselves with the idea that if one got less than 10% reporting of "dreaming" from NREM wakings and over 80% from REM wakings then dreaming occurred in REM but not NREM sleep, Foulkes (1962) examined recall in all stages of sleep without ignoring "thinking" reports. He found over 50% of "dreaming" content in all stages of sleep - including stage 4. But he found recall of some content, whether thinking or dreaming, in three-quarters of all NREM sleep wakings. He found significant qualitative differences between the type of mentation in REM and NREM sleep, in the rate of activity, emotion, number of scenes, and distortion, for instance. These differences could be summarized by saying that NREM mentation was less dramatic than REM mentation. But he suggested that "reportable mental activity is always present in the sleeping human".

Rechtschaffen et al (1963) sought to examine these differences again. They reported that "thinking" rather than "dreaming" (subjects' own judgements) was commoner in NREM sleep; recall in NREM sleep was described as "poorer". In NREM sleep fewer experiences were described as vivid, fewer as primarily visual (though 40% were so described), fewer as bizarre, fewer as emotional. There was more volitional control in NREM mentation, more experiences were called pleasant, more were/

/were related to the subject's immediate life. Problems of bias on the part of both experimenters and subjects are more acute in studying NREM than REM recall (Herman et al 1970). Nevertheless, although Rechtschaffen et al regarded Foulkes' interview technique as too liable to elicit confabulatory material, their findings are substantially in agreement. Rechtschaffen et al concluded that NREM mentation resembles "that large portion of our waking thought which wanders in seemingly disorganised, drifting, non-directed fashion whenever we are not attending to external stimuli or actively working out a problem ..."; they also noted that although memory for NREM experience seems poor, our memory for waking thought (other than problem solving) is in fact equally poor, although our memory for visual experiences in waking is quite good.

These observations as to the differences between REM and NREM mentation have been generally confirmed by later workers (Goodenough et al 1965a; Foulkes, 1966; Pivik and Foulkes, 1968), although Kales et al (1967) for instance, using a relatively strict definition of dreaming, minimise dream recall from NREM sleep. Monroe et al (1965) showed conclusively that REM and NREM reports were discriminable by blind judges familiar with the reported differences in mentation. Correct discrimination was achieved on 70% of reports by an automatic separation of reports with content as "REM", reports with no-content as "NREM". Separating reports into primarily perceptual or conceptual yielded 74% success. Discrimination by the judges gave 80% success using all reports; matching pairs of reports for subject, night and time of night improved this to 89%; when no content reports were/

/were excluded success rose to 92%, and using only those the subject called a dream they achieved 94% correct discrimination.

I have just been stressing differences between REM and NREM mentation. It is now generally accepted that some form of mentation is common at least some of the time in NREM sleep. But it is less widely accepted that subjects dream outside of REM sleep. Nevertheless Foulkes and Wögel (1965), studying sleep onset hypnagogic mentation, concluded that hypnagogic content can be as regressive and symbolic as REM dream content; in a majority of cases subjects hallucinated their own participation. Even neglecting hypnagogic processes, long recognised as having dreamlike qualities (Maury, 1865; Oswald, 1962), Foulkes (1967) has pointed out that "the modal NREM experience is not described by subjects as a "thought" but as a "dream", i.e. it is ... an hallucinatory, visual experience...". Zimmerman (1970) studying differences between light and deep sleepers' mentation in both REM and NREM sleep found that while his group of deep sleepers said they were dreaming more often when woken from REM than NREM sleep, his light sleepers did not discriminate REM and NREM mentation in this way. Nor did they discriminate between REM and NREM mentation on the dimension of perceptual versus conceptual experience. He found that there were, however, dimensions which discriminated REM from NREM mentation in both light and deep sleepers even when NREM mentation was regarded as dreaming. These were the degree of active participation, thematic continuity, emotion, clarity, vividness, dramaticity, degree of recall and amount of physical activity. Thus it would appear on balance that there are variables which will distinguish REM from/

/from NREM experiences, but these do not necessarily include whether the subject regards himself as dreaming, nor whether the experience is primarily visual.

7. Nightmares and REM sleep:

Nightmares can occur in both REM and NREM sleep, but they take different forms. Nightmares in NREM sleep (night terrors) involve an abrupt arousal, usually from stage 4 sleep. In persons susceptible, NREM night terrors can be provoked by for instance sounding a buzzer. The intensity of such terrors seems to be related to the duration of the preceding stage 4 sleep - the longer it has been continuing, the more intense the terror, as judged by the accompanying increase in heart rate (Broughton, 1968 ; Fisher et al 1970a). NREM terrors are quite rare in the population - only 5% of subjects complaining of nightmares are subject to these. Content associated with the sudden arousal and terror is minimal, perhaps involving some one image or scene.

REM nightmares (or anxiety dreams) show quite different features. In NREM terrors there is no autonomic variability prior to the arousal, at which point huge increases in heart rate are observed, often reaching 120 a minute. By contrast, in the REM anxiety dream, there may be some autonomic indication during the REM, including vocalisation (Evans and Oswald, 1966). When (if) the dreamer awakes, there is then only a relatively slight increase in heart rate - say 20 beats per minute. Fisher et al (1970a) have described an increase in heart rate from 60 to 70 and respiratory rate from 18 to 24 per min along with a rise in the profusion of eye movements in the terminal minutes/

/minutes preceding three severe REM anxiety awakenings.

Autonomic functioning returned to normal soon after the arousals (in contrast to NREM arousals). However, Fisher notes that in many quite severe (high anxiety) nightmares in REM sleep there is little or no autonomic accompaniment to the subjective fear. Fisher et al (1970b) describe how in 22 spontaneous wakings with anxiety from REM sleep, 18 of these were associated with heart rates and respiratory rates which were no different from those of the same (eleven) subjects in REMPs without anxiety wakings. Fisher et al discuss what they term the "desomatisation" of anxiety in REM sleep nightmares. Emphasising the frequent dissociation of the psychological and the physiological accompaniments of anxiety in nightmares, they regard the REM nightmare as the "controlled" anxiety dream; the desomatisation may help to keep the dreamer asleep - but if the anxiety becomes too great, it will "break through" - high levels of autonomicall activity and spontaneous waking will follow.

If Fisher tends to emphasise the lack of psychophysiological parallelism in the nightmare, Hartmann (1970) stresses the correlation between factors which tend to increase REM sleep - apparently for other than psychological reasons - and those same conditions leading to nightmares. Hartmann notes that in adults nightmares are rare except in pathological conditions and after drug use. Most of the situations he describes, he associated with high REM time. First, there is the observation that nightmares are frequent in childhood, but not in adulthood. The amount of REM sleep (both proportionately and absolutely) taken by children is somewhat greater than that/

/that taken by adults. However, it is also worth noting that nightmares have never been noticed during recovery from REM deprivation (by awakening) although REM time is high on recovery nights.

The one case of a nightmare occurring in a normal subject without medication reported by Hartmann was that of a young woman who woke from a 25 minute REMP, a day before the onset of her menstrual period, when REM times are generally high, as they were in this instance (32% for the night - the normal range being 20-25%). Hartmann also reported that on this night her eye movements were unusually profuse. He reported also on two other cases where manic-depressive patients had nightmares in REMPs lasting between 20 and 50 minutes which were very plentiful with EMs.

Nightmares are also reported by people recovering from fever. Karacan et al (1968) induced experimental fever for one night and demonstrated that this reduced REM times drastically. Though there was no recovery "rebound" in their study, this remains a possibility after several nights of REM deprivation by fever and/or associated medication outside the laboratory.

Such instances should perhaps be subsumed under those nightmares which have been reported during drug withdrawal. Hartmann quotes several cases from his own laboratory of nightmares two or three nights after the withdrawal of sleeping pills. Kales and Jacobson (1967) and Kales et al (1968a) have also described such nightmares in patients withdrawing from pentobarbital on which they had been dependant, and even after one woman had taken Noludar (methyprylon)

/(methyprylon) 300mg for only three nights. They also reported "occasional nightmares" after pentobarbital, methyprylon and glutethimide had been taken by normal subjects for only three nights (Kales et al 1970).

Oswald and Priest described nightmares in both of the subjects in their experiment on withdrawal from 600mg amylobarbitone, when nearly half of their total sleep was spent in REM. Interestingly, nightmares were not complained of after withdrawal from nitrazepam. This may have been because the dose was relatively less, but could also be because the drug is probably eliminated more slowly, producing less sharp a peak in REM rebound. Le Gassicke et al (1965) also reported nightmares in a patient withdrawing from tranlylcypromine, an amphetamine derivative: the patient awoke from one sleep onset REMF uttering blood-curdling shrieks.

Greenberg and Pearlman (1967) mention the occurrence of a nightmare in a patient withdrawing from a period of dependence on alcohol. Gross et al (1966) also report nightmares from persons still on heavy doses of alcohol. While this might seem at variance with the cases so far mentioned, since alcohol suppresses REM sleep, it will be recalled that Yules et al (1966) showed that an increase in REM sleep can develop during repeated alcohol administration.

Lehmann and Ban (1968) also reported a nightmare in one of their subjects after taking nitrazepam 10mg for only one night; on active drug nights their subjects reported less dreaming with nitrazepam, which is consistent with the occurrence of a withdrawal nightmare. (Surprisingly, however, their subjects reported dreaming more on/

/on quinalbarbitone).

Nightmares can also occur with drugs which increase REM sleep following administration. They have been reported following reserpine (Wilkins, 1954; Winsor, 1954), which has been shown to increase REM time (Hartmann, 1966; Coulter et al 1971). Further, Evans and Oswald (1966) reported 5 occasions among 2 dozen nights when narcoleptic patients given 5 grams tryptophan appeared to be experiencing nightmares - emitting strangled utterances suggestive of internal torment. Giving tryptophan to narcoleptic patients was shown to roughly double the length of their (typical) sleep onset REM, and also increase the number of EMs per minute (the tryptophan and control records could be reliably discriminated on this account, according to Evans and Oswald, 1965). Lewis and Oswald (1969) reported that nightmares occurred in all three of their patients recovering from overdoses of tricyclic anti-depressants. These (female) patients all had frightening dreams with sexual themes about a dozen nights after taking the overdoses, at the time when REM sleep was most prolific. Lewis and Oswald comment that the sexual content of the dreams was probably a "true withdrawal phenomenon", since nocturnal erections are linked with REM sleep, and so are nocturnal clitoral erections (Karacan et al 1970). Lewis and Oswald stressed the association between the occurrence of nightmares at times of drug withdrawal (including the time of peak withdrawal of the drug from brain tissue after overdose) and the relatively great profusion of eye movements also occurring at this time (when REM sleep is enhanced).

However, it may not be the high profusion of eye movements which is the crucial variable associated with withdrawal nightmares. Those drugs which produce nightmares on withdrawal are in general anxiolytic to some extent, and it may be a withdrawal "rebound" in anxiety which is crucial in producing nightmares - or a combination of high "REM pressure" and high anxiety. Kales et al (1969b) demonstrated high self-rated anxiety on withdrawal from REM suppressant drugs. Dr. O. Ogunremi has recently completed some experiments in this laboratory on the effects of a minor tranquilliser (benzocetamine) and sodium amylobarbitone on daytime anxiety and concentration levels and plasma cortisol. Both benzocetamine and amylobarbitone reduced daytime anxiety and concentration, and also night-time plasma cortisol levels. Withdrawal of both drugs produced a "rebound" increase above baseline levels of daytime anxiety and also a rebound above baseline in levels of night-time plasma cortisol. Such clear evidence of high anxiety levels following drug withdrawal in normal subjects, at a time when nightmares may occur, suggests that high anxiety may be at least as important an element in the occurrence of nightmares as high profusion of eye movements or high "REM pressure".

CHAPTER THIRTEEN

THE EFFECT OF DRUGS ON THE CONTENT OF DREAMS

Reference has just been made to the frequency of nightmares in drug withdrawal. Studies of the effect of drugs on "dreaming" are frequent - usually referring to no more than the effect of a drug on the percentage of REM sleep in the night. Studies on dream recall frequency, or more especially on the content of dreams under drug administration, are few. To instance one of the former studies, Shaskan et al (1970) produced a paper entitled "Does Noludar (methypylon) change dreams?". Starting from anecdotal reports from their patients that they dreamt less while on methypylon, the authors finally concluded "fewer pleasant dreams were admitted during the drug phase, but on follow-up it was also found that fewer unpleasant dreams were admitted during this phase". (!).

1. Studies by Whitman and others

Whitman et al (1960) were the first to use EEG techniques to examine the effect of a drug on dream content in a systematic manner. They used one patient who received eight nights without any medication, followed by nine nights with 400mg of meprobamate. The authors remarked that they were unable to conduct a doubleblind study because of the characteristic changes produced in the EEG. The patient was woken after 5 minutes REM sleep, and given a structured interview. The report was taped, and scored on a battery of scales for hostility, anxiety, dependency, heterosexuality and homosexuality, intimacy and motility. Saul and Sheppard's hostility scale (1956) was used as a model; they devised the rest following the same format. They/

/They scored the manifest content of the dream, but "also made free use of the associations (derived as part of the interview) .. and the connotations, as well as stated meanings, of various words. " We decided that it was an injustice to the richness of the manifest dream if we did not treat it as a complex, symbol-laden, condensed psychological product". Three judges indepently scored the dreams on all the scales (the scoring was "statistically reliable"). They noted that the dreams before and during medication could not be distinguished from each other; detailed scoring of the dreams was necessary to reveal any effects. These were limited to significant increases in motility and dependency on the drug.

Whitman et al (1961) extended their examinations to phenobarbitone (10mg), prochlorperazine (5mg), and imipramine (25mg). They were administered double-blind in a balanced design. Each volunteer subject (10 in all) first had one night without medication; this night was not included in the balanced design, and appears to have been the subjects' first night in the laboratory, so that unfortunately this night is not comparable with the other nights, because it is subject to the 'first-night effect'. Among other things, the frequency of no content reports following REMP wakings may be lowered on the first night of dream collection (Firth, 1972). Each of the three drugs was administered for three days, with dream collection on the third night. There were four-day intervals between periods of drug administration (Whitman, 1963). The scales used by Whitman et al (1961) were again used on the manifest content of the dreams, though again the scorers were "alert to symbolic or other allusions to/

/to important affective material". The inter-rater reliability was reported as .75. The number of dreams reported was significantly lower on imipramine than on baseline; this appears to have meant that the number of REMPs from which wakings were made was reduced, since the procedure was to wake the subject from all REMPs after 5 minutes of REM sleep. The scores on the various scales were significantly lower on imipramine and prochlorperazine than on either baseline or phenobarbitone, though these differences disappeared when the scores were corrected for word length. Phenobarbitone increased the score per word for homosexuality, and imipramine the score per word for anxiety at barely significant levels ($p < .10$); hostility per word was significantly increased by imipramine ($p = .02$). In their discussion the authors note that they regard the hostility and anxiety scales as over-lapping considerably. In terms of explanation for the effects they found, they say that imipramine "might be seen as a hostility mobiliser which results in hostility discharge during the dream state" and phenobarbitone increases the expression of homosexuality "since it is a general sedative and increases passivity". The rather loose generality of such conclusions would seem partly to be a consequence of the fact that the authors did not set out with any theoretical formulation or any empirical basis for predicting the effect of the drugs.

Whitman (1963) discussed some further findings from the same experiment, relating to the intrusion of the experimental situation into dreams (and also in another study using patients, the question/

/question of which dreams are told to the therapist the next day). Whitman noted that of 111 dreams, 36 dealt in an undisguised way with the experimental situation, and 40 more dealt with it in a disguised fashion. These elements took the form of anxieties about various aspects of the situation. The experimenter (male) was seen as seductive by the women, sadistic by the men; anxiety was manifested over possible loss of communication, abandonment, or being left in isolation; anxiety was expressed about being damaged by the equipment, and overall the authors gained the impression that a superficial compliance frequently hid deeper anxieties about the experiment.

2. A study by Kales and others:

Kales et al (1969b) examined the effects of glutethimide and methyprylon on sleep and on dream content. Glutethimide 500mg and methyprylon 300mg were chosen as REM suppressant drugs (Kales et al 1968a). Uninterrupted sleep studies were done in the laboratory, and dream recall studies were done at home; the intention being to collect sleep data without disturbing sleep for dream recall. The dreams were thus only derived from morning recall, not from REM interruption. However, the subjects' maximum total sleep time was controlled to 8½ hours by alarm clocks. The drugs were administered for three days twice with four days of placebo between the two administrations. Three days of placebo also preceded the first administration and followed the second. Six subjects were studied with each drug. The rationale of giving the drug in two separate periods of three days was not clear, though one supposes it was intended to provide a/

/a replication for each subject. Such a design seems to fall between the two stools of the single night study (which prevents tolerance developing), and a six night study which could evaluate tolerance effects and the withdrawal effect which was one of the authors' chief interests, since they had previously noted nightmares in withdrawal from these drugs. Dream recall frequency was assessed by the subjects (how many they felt they had had each night), and by the experimenters (the number with some specific content). The experimenters also rated the dreams on a three-point scale for detail, and unpleasantness. It was not stated whether the dream ratings were done blind or not.

Both drugs decreased dream frequency, but withdrawal frequencies were no higher than baseline. Dream detail was also decreased by both drugs, as was dream unpleasantness. Withdrawal from both drugs produced greater dream unpleasantness than baseline, but greater dream detail on withdrawal only occurred with glutethimide. Glutethimide produced more consistent and greater changes than methyprylon; this would appear to tie in with the fact that tolerance to the REM suppressing effect of methyprylon develops within three nights of drug administration, so that effects averaged over three nights would be expected to be less striking than with glutethimide. Three nightmares were reported by the subjects, one being "the worst nightmare of my life". Mood was also evaluated during the EEG sleep study (apparently using different subjects), by an adjective check list. This revealed that anxiety was highest during withdrawal of both drugs (though it was not necessarily lowest during the active drug phase), and depression was lowered by/

/by the active drugs and raised on withdrawal. This latter trend was the only one to prove statistically significant. Although the mood results were evaluated statistically, it seems unfortunate that the dream ratings were not.

3. A study by Carroll and others:

Carroll et al (1969) hypothesised that since barbiturates (among other drugs) reduce not only the amount of time spent in REM sleep, but also the profusion of EMS within REM sleep, and since profusion of EMS had been related to the "activity" of the accompanying dream, then barbiturates should make dreams more "tranquil" and "passive". They gave 200mg amylbarbitone to three subjects. Each came for one adaptation night, which included REM sleep wakings, and then three further nights. On the first, subjects received placebo; four days later they got active drug, which they took for a further 6 nights at home; on the last night (the first withdrawal night) placebo was again given. Wakings were made after 10 minutes of the second, third and fourth REMs, and the subjects' reports were collected in accordance with the standard interview of Foulkes (1970b). These reports were then scored by two judges, blind as to the conditions of each report, on Foulkes' dreamlike fantasy scale (Foulkes et al, 1966), and as "active" or "passive". One of the raters also noted instances of sexual content. It was found that the scores on the Foulkes scale were significantly lower on active drug than either on baseline or withdrawal, though these two conditions were not different. Three dreams occurred which were sexual in content: all occurred on withdrawal. "Passive" dreams were/

/were significantly more probable on drug than on placebo.

These effects were not ascribed to a failure of recall on barbiturate, on the grounds that in the waking state barbiturate had been found to facilitate recall (Batten, 1967). Batten in fact found a trend for ~~recall following~~ 100mg phenobarbitone (versus 10mg amphetamine) to enhance recall over a period of up to 20 minutes, and depress it after 1 hour or more, but these differences were not significant. Although the time subjects took to wake and respond was not recorded, it was the authors' impression that subjects took no longer to respond in the barbiturate condition- it has been shown that gradual arousal can increase the probability of obtaining a "thinking" report rather than a "dreaming" report (Goodenough et al 1965a; Shapiro et al 1965).

Carroll et al concluded that the barbiturate had made the dream experience "more conceptual and less perceptual, more "thought-like" and less "dream-like". The Foulkes' scale actually examines several 'dimensions': perceptual/conceptual, Visual/non-visual, hallucinatory/non-hallucinatory (real at the time, or not), and bizarre/everydayish. Inspection of Carroll et al's published data in fact reveals that none of the barbiturate dreams were rated by the subjects as real at the time, and this is where the difference between drug and placebo conditions arose. More accurately they should have concluded that barbiturates make the dream more "passive", and less real at the time.

4. A study of dream recall at home by Morgan and others:

Another study performed since has also examined the effect of 200mg amylobarbitone, as well as dichloralphenazone (Welldorm) 1.3g, nitrazepam (Mogadon) 10mg, methaqualone 250mg + diphenhydramine 25mg (Mandrax), and/

/and placebo (Morgan et al, 1970). This study, like that of Kales et al (1969b), used home dream recording, with the consequent loss of material as compared with a REMP interruption technique. Treatments were double-blind, based on a Latin square design. Two nights were kept free of drugs between experimental sessions; subjects were not allowed tea, coffee, alcohol or other drugs after 7.00 p.m. on the experimental nights. Unfortunately, they were not kept free of alcohol at other times. Since alcohol taken in the early evening can almost certainly affect REM sleep that night, this degree of control over other drug use should not be regarded as sufficient, as subjects rarely happen to take identical amounts of alcohol at the same time each night. Subjects were woken each morning at their time of usual waking by telephone. The dream reports were taped, and specific questions asked at the end of the spontaneous report to prompt the subjects' memory for further dream material.

Dreams were analysed for hostility and anxiety according to the scales of Gottschalk et al (1960) - these scales are similar to those used by Whitman et al referred to already. The number of dreams recalled was significantly lowered on amylobarbitone, but was not affected by the other active drugs. On amylobarbitone, those dreams which were recalled contained no anxiety or hostility. Nitrazepam also reduced the expression of hostility. Dichloralphenazone apparently increased anxiety in the dreams. These results could not be tested for significance owing to the number of subjects not recalling any dreams - no dreams were recalled on 36% of subject-nights.

The authors themselves point out that the effects of amylobarbitone may well have been due to poor recall of dreams as much as to any effect of the drug on anxiety or hostility - especially since only 6 dreams were obtained under this condition. The authors were modest about the scope of their study. Nevertheless it should be borne in mind that in home studies factors affecting dream recall are necessarily confounded with factors affecting the content. A hypnotic drug may reduce the number of dreams recalled both because it may depress the quantity of REM sleep - and so reduce the probability of being woken from a REMP in the morning - and also because it may reduce the chance of waking spontaneously near the end of a REMP during the night. But it may also selectively affect recall of those dreams of which some content is recalled. Orr et al (1968) have shown that some subjects at least are capable of waking themselves from REMPs under certain conditions. Salamy (1970) has also shown that behaviour in REM sleep can be altered by reinforcement. It would seem that in a situation where subjects are being asked about their dreams (and are very probably receiving some reinforcement, however unintentional, from the experimenters for recalling dreams) some of them may wake themselves from REMPs. It is certainly my own experience that one can 'learn' to wake almost at the end of every REMP if one wishes to recall dreams. Given these factors, sedative drugs may affect the speed of reaching sleep after a brief waking during the night, and may differentially affect the recall and rehearsal of material during this period, especially if the dream material is

/is emotionally loaded. I would concur with Morgan et al, despite these remarks, that useful information may be obtained from subjects in their home environment, provided it is recognised that home studies and laboratory studies should be used for different purposes.

5. A study by Deichsel:

One other study involving the effects of a hypnotic on dream content has recently been made. Deichsel (1972, in press) examined the effects of methaqualone and diphenhydramine (Mandrax) on the dreams of extroverts and introverts. 29 subjects were categorised as strongly introvert, strongly extrovert, or neither. Subjects in the first two groups either took the hypnotic daily, or took no medication. Subjects' dreams were collected from REMPs on two nights, and from NREM sleep on the third night. There were apparently no differences between REM and NREM dreams, which seems curious in view of the large number of other reports that such differences exist. Dreams from all the wakings were scored on rating scales for anxiety, aggression, and activity of the dreamer. The frequencies of familiar and strange characters, animals, colours and sex symbols were noted, and the incidence of "positivity" and of laboratory dreams was recorded. Certain differences between the dreams of introverts and extroverts were noted: essentially extroverts' dreams contained more activity, anxiety, aggression and sex symbols, and more characters.

The hypnotic apparently affected the frequency of dream reports to a greater degree with the extraverts than with the introverts, the/

/the frequency of dream reports being lowered in both groups. No information is given as to whether this reduction reflects a smaller number of REMPs from which wakings could be made, or whether it reflects a smaller percentage of recall from awakenings. Apparently dream content of both introverts and extroverts on drug resembled introverts' rather than extroverts' dreams; one presumes therefore they contained less activity, anxiety and aggression, and fewer characters and sex symbols. It would appear from this paper that the dreams of introverts taking the hypnotic do not differ much from those who took no medication, while the dreams of extroverts on drug were strikingly different from those of extroverts not taking the hypnotic.

Assessing this report is difficult. It suggests that the hypnotic may alter dream content, at least in extroverts, by reducing factors such as activity of the dreamer, and the number of characters and the degree of aggression in the dream. It does suggest that the effects of a drug may interact with personality. Unfortunately the results are only presented in descriptive terms; no quantitative material is offered. The lack of differences between REM and NREM reports even in subjects not taking the drug, for which no explanation is offered, and the lack of detail about the experimental procedure, make one rather sceptical of the results as a whole. In particular, it is not clear to what extent the results reflect differences in the frequency of reports with some content, or differences within the content. This especially applies to the comment that there were no differences/

/differences between the three nights (respectively REM, REM and NREM awakenings). Some statements also appear to contradict others - at one point it is stated that "We did not find well-established differences between the groups unless the subjects were separated according to the condition 'with vs. without hypnotics'". This would appear to be inconsistent with other statements that clear cut differences between extraverts and introverts were lessened when subjects taking the hypnotic were compared. There are other points where we lack information, as on the number of subjects in each of the various experimental groups.

Another disappointing feature of Deichel's study is that between-subject effects were apparently confounded with drug effects per se. Some extroverts took the hypnotic; some did not. No mention is made of whether subjects taking the hypnotic differed in any way from those not taking the hypnotic; it is possible that differences between these groups might have been responsible for the apparent effects of the drug. It would have been preferable if some subjects had experienced both drug and non-drug conditions in a balanced, controlled design.

CHAPTER FOURTEEN

THE INTENT OF THE PRESENT STUDY

This experiment was conceived as replication and extension of that by Carroll et al (1969) already described. Carroll's experiment involved only three subjects, being recorded on only one night each under each condition. Furthermore, Carroll et al examined the dream content only in an overall fashion, that is, they used ratings for the whole dream. These ratings comprised, in effect, a series of dichotomous categories - was the dream active/passive, sexy or not, bizarre or everydayish etc. It was hoped that by looking at the dream in more detail - by using content analysis - more information could be gained. In particular it could be established whether the effects of the hypnotic were in terms of visual activity solely, (i.e. whether there were no other effects), or whether the sedative effects of the drug extended to activity generally, and to such features as the number of participating characters, whether interactions were aggressive or friendly, and whether the effects would be evident solely in the manifest, or also in the latent content of the dream (using the scales of Whitman et al).

1. Hypotheses:

It will be recalled that Carroll et al's rationale for their experiment was the effect of the barbiturate on eye movement profusion, given some association between EM profusion and dreaming. Here it was hoped first to test a prediction from the scanning hypothesis - would the reduction in EM profusion be associated with a reduction/

/reduction in the visual aspects of dreaming. Would barbiturates make dreams specifically conceptual rather than perceptual? This can be tested quite simply using Foulkes' dreamlike fantasy scale.

Secondly, it was hoped to test the intensity hypothesis. Given that the active drugs reduced the measure of REM intensity - EM profusion - it was predicted that dream content would involve less active participation by the dreamer, a reduction in the number of activities in the dream, a reduction in the number of characters, in the number of interactions between them, and in the number of dreams involving overt emotion.

A third, very specific hypothesis was made from the assumption that the distinctive features of REM as opposed to NREM mentation are associated with the presence of EMS in REM but not NREM sleep. To the extent that the active drugs suppressed the EMS, it was predicted that dream content would resemble NREM mentation. Foulkes' dreamlike fantasy scale (Foulkes et al 1966) was originally devised to discriminate REM from NREM reports, using not just the distinction between visual and non-visual mentation, but also the "reality" and bizarreness dimensions, and it was therefore ideal for this purpose.

A fourth hypothesis emerged during the study. It had been expected that barbiturates, having sedative properties, should reduce anxiety in the dream content. The independent judge (Ian Oswald) who rated the dreams as active or passive, and for sexual content, chose also to rate them for anxiety. Anxiety and hostility scales were also chosen from Whitman et al (1961). When the dreams were/

/were passed to one other independent judge (John Trinder) for scoring on the Hall - Van de Castle system of content analysis, he formulated a more general "quality" hypothesis: sedatives should make the dream experience pleasanter. Drug dreams should contain more emotion of happiness, fewer of anger, apprehension, confusion, sadness. They should contain relatively more friendly interactions, and fewer aggressive ones. They should contain more success and good fortune. Vice versa, on withdrawal. Such a hypothesis can also be made on the basis of Kales' et al (1969b) finding that their hypnotics decreased dream unpleasantness, and increased it on withdrawal.

2. A second hypnotic:

It was also felt worthwhile to extend the study to include a non-barbiturate hypnotic. Nitrazepam (Mogadon) seemed suitable, as a minor tranquillizer. It too affects the profusion of eye movements in REM sleep. If the action of these drugs on dreams were solely, or largely, in terms of their effects on the eye movements, then their effects on dreams should be similar, and in proportion to their EM suppressant effects. As will be seen from the results, at clinically equivalent doses, nitrazepam has if anything a greater EM suppressant effect than does amylobarbitone.

Nitrazepam has been shown to be a safe (Matthew et al 1969) and effective hypnotic in doses from 5-10mg (Baum et al 1965; Tetreault et al 1966; Hadier, 1968). 5mg and 10mg nitrazepam would seem to have effects comparable to 100 and 200mg amylobarbitone both clinically (Davies and Levine, 1967; Haider, 1968) and in terms of its effects on sleep, and in particular REM sleep latency and amount (Haider and Oswald, 1971). Haider and Oswald also showed that at these doses their effects in reducing spontaneous electrodermal activity during REM sleep were similar: in so much as this can be used as a measure of their anxiolytic effects, then, they are comparable.

However, it has been argued that the mode of action of the two drugs is radically different. Whereas barbiturates appear to exert/

/exert their sleep inducing effect by depressing both cortical and subcortical structures including the brain stem reticular activating system, evidence has been presented from which it is speculated that nitrazepam acts chiefly on the limbic system (Takeshima, 1971). Thus Soulaire et al (1965) showed that in the rat and in the rabbit reticular formation activity was only slightly depressed in comparison to the effect of a barbiturate. This study did not present numerical data on this effect, however. More important, the doses used were exceptionally high relative to normal clinical doses (10mg/Kg, equivalent to about 500mg nitrazepam in an adult human). Gogolak and Pillat (1965) showed that the hippocampal arousal threshold in the rabbit was greatly raised by nitrazepam, which only slightly raised the cortical arousal threshold. The action of a barbiturate was the reverse. These authors did present numerical data; their doses (0.8 to 2.5mg/Kg nitrazepam and 5 to 10mg/Kg pentobarbitone) were nearer human clinical dose levels. The doses in Takeshima's study were directly equivalent: he gave 0.1mg/Kg to cats, equivalent to 5mg in an adult human. The inference from these studies is that nitrazepam acts as a hypnotic partly through its action on limbic structures and only to a lesser degree via its depressant action on the reticular formation. Barbiturates by contrast are supposed to be effective largely through their effect on the reticular formation.

Both barbiturates and nitrazepam affect the EEG for at least 12 hours after even a single dose (Walters and Lader, 1971). However, nitrazepam acts more slowly than amylobarbitone in its effects on the EEG, although some effect is evident within 10 minutes of oral/

/oral administration (Volovka et al 1969). Nitrazepam's effect on the EEG was still detectable after 5-10mg up to 18 hours later, when significantly more drowsiness was detectable than after 100-200mg amylobarbitone (Malpas et al 1970).

Since the time course of its action is longer, nitrazepam would be expected to show differences from amylobarbitone in its day by day effect on sleep and on dreams: in particular, the peak of the REM sleep rebound on withdrawal occurs after about three days of nitrazepam withdrawal, whereas after amylobarbitone the peak is reached within one, or two, days (Oswald and Priest, 1965). In this experiment, however, differences in the effects of the two drugs were not anticipated as a result of this variable, because the sampling nights were chosen to select as close as possible the nights of maximal effect. Thus by recording the second withdrawal night, withdrawal effects of both drugs would be expected to be near maximal.

That the mode of action of nitrazepam is as a tranquillizer rather than via a direct effect on mechanisms maintaining wakefulness enables discriminating predictions to be made on the comparative effects of the two drugs on dream content. If the two drugs' action on dream content were solely a result of their action on EMS, then no differences would be expected between the two drugs in their effects on dream content. This would be predicted on the scanning hypothesis, the intensity hypothesis, and on the hypothesis that reduction of EMS would make REM mentation like NREM mentation. By contrast, a "quality" hypothesis would predict differing effects of the two drugs on dream content, to the extent that it is possible to discriminate between tranquillizing/

/tranquillizing and c.n.s. depressant effects on mental processes.

3. The two experiments:

In the first experiment, two doses of each drug were used: 200 and (after one week) 400mg of amylobarbitone, and 10 and 20mg nitrazepam. The design (to be discussed in Chapter 16) was for one week on the low dose, followed by one week on the high dose, with data collection nights on the first night of each dose, and after one week on each dose, besides on withdrawal. This allows any dose, and tolerance effects to be evaluated.

After the first experiment was completed, it was thought desirable to verify the results with the use of more subjects. For this experiment, only amylobarbitone was used, and dreams were only collected on the initial night at each dose, since no tolerance effects were evident from the results of experiment 1.

I shall now proceed to review some aspects of dream content analysis.

CHAPTER FIFTEEN

THE DESCRIPTION OF DREAM CONTENT

Qualitative descriptions of dream content are not new.

The most famous of these is Sigmund Freud's, *The Interpretation of Dreams* (1932). Another excellent qualitative approach to dreaming is that of Calvin Hall (*The Meaning of Dreams*, 1966). More recent qualitative approaches focussing on specific questions are those of Kramer et al (1964) - they focussed on the interrelationship of dreams within one night - and Witkin and Lewis's study (1967) of the effects of pre-sleep experiences on dream content. An approach which foreshadowed later quantitative methods of 'content analysis' was that of Eggen (1952) who classified the frequencies among some 250 dreams of the Hopi of various themes such as physical hazard, heterosexual elements, elements of persecution and conflict, and emotions.

Quantitative approaches to the content of dreams can take various forms. A rather special one was used by Berger (1963) - he got his subjects, and independent judges, to try and match dreams to the stimuli which had been presented during sleep; he then calculated the chance probability of getting the number of correct matches obtained. Clearly such an approach is developed to solve particular problems. The principle however is of more generality. If I have dreams from a variety of different conditions, and I wish to establish whether there really are differences between the dreams, or whether I am merely deluding myself, I can give the dreams to someone else who has never seen them before, tell him what I predict, and see/

/see whether he can correctly guess which reports came from which conditions. Such an approach was used by Monroe et al (1965) in deciding whether REM and NREM dreams were discriminable. I have chosen to use it in this study too.

This procedure tells you whether two groups of dreams are different, but it does not tell you how they differ. (The judges will no doubt tell you, but they may be unaware of all the factors which influenced them and especially if they have some vague, pre-conceived and theoretical ideas about what differences they expected, they may tell you some things they bore in mind which might not be the crucial variables).

The problems then is to try and see if from one's own hunches or other peoples' it is possible to quantify measures of content in a reliable fashion. The validity of scoring procedures can only be established by reference to outside variables. For instance, the psychodynamically oriented scales of Whitman et al (1961) were recently used in an unpublished study in this laboratory by Maureen Burns and Stuart Lewis on the effect of the menstrual cycle on dream content. Two women were recorded weekly over a period of months to sample their dreams at various points in the menstrual cycle. The sexuality scales used interpretations of latent sexual content as well as of manifest content. The fact that variations in the sexual content were found over the cycle (with peaks at the menstrual and ovulatory periods) argues post-hoc that the scales had some validity, provided one can assume that they were reliable.

Reliability can be established in essentially two ways. First, does one rater give the same scores to the same dreams if he scores them on two occasions separated by an interval of time (test-retest reliability)? It would be possible to test reliability this way without the rater ever having to make explicit his criteria for scoring a dream. More usually, the originator of the scale writes down his criteria and then gets someone independent to see if he can reach the same results using only the stated criteria (inter-rater reliability). It is in this form that reliabilities for most dream content scales are presented. However, it is necessary to bear in mind what the stated reliability refers to. It is possible to give reliabilities for each element of a dream, for the whole dream, or for a subject's mean score. For a given scale the first may often be quite low, especially if the scale involves many categories, whereas reliabilities for means are usually very high. Moses et al (1972) discuss this problem in relation to reliabilities for sleep stage classification.

Quantification of dream content can be done on two kinds of items. It can be done for the dream as a whole - usually by rating the dream on one or more dimensions - or it can be done on elements within a dream. The latter approach particularly is known as "content analysis".

Quantification may also be done on three levels. Categorisation may be nominal. Items are assigned to one of several categories (e.g. male or female). There is no implication that one is/

/is 'greater' than another. Alternatively, items may be assigned to one of several categories which are considered to be in some order of magnitude. Most rating scales fall into this group: the scale is ordinal. A scale for novelty might be constructed thus: highly familiar, not familiar but encountered before, never encountered but would not be unexpected, never encountered and would be very strange. Thus it could be said that one item was more or less novel than another. Both of these levels of analysis can yield frequencies - e.g. the number of familiar items in a given dream.

An interval scale of measurement implies that ratios can be specified between points on the scale. Thus it is not possible to say that a strange and unexpected object is twice as strange as one which is unfamiliar but not unexpected. On the other hand it is meaningful to say that eight people are twice as many as four people. Such scales therefore deal in intensities, although they may be utilised to provide frequencies (the number of dreams with only one character).

Some dream content studies use the subject as the judge, with or without other judges. One of the easiest ways to gain some information about the dream is to ask the subject a series of yes/no questions (a simple nominal categorisation). Such questions are often used to supplement other methods of analysis, or to provide data which may be useful for some other judge in rating aspects of the dream - as has been done in this study. Such questions can be/

/be included at the end of an unstructured interview, or may be used as the bulk of the interview, as in the Foulkes dreamlike fantasy scale (Foulkes et al 1966) in which answers to a series of yes/no questions are used to rate the dream into one of eight categories. For theoretical reasons the scale is regarded as ordinal, but it has been treated both purely as a nominal scale (Carroll et al 1969) and as an ordinal scale (Foulkes et al 1966; Pivik and Foulkes, 1966, 1968).

One study which made efficient use solely of yes/no questions asked of the subject was Rechtschaffen et al's study (1963) of mental activity in REM and NREM sleep, referred to in Chapter 21. Another study of mentation in REM and NREM sleep which relied heavily on yes/no questions was that of Zimmerman (1970), also referred to in Chapter 12. The subjects in Zimmerman's study also rated their experiences on various ordinal scales, for dramatic quality, distortion, vividness, etc.

Dichotomous or yes/no ratings for the whole dream have also been employed by experimenters, as in the studies on active and passive dreaming by Dement and Wolpert (1958), Berger and Oswald (1962) and Carroll et al (1969). Yes/no ratings have also been used to score for the presence or absence of an element within the dream, as in Beck and Hurvich's study on the frequency of masochistic elements in the dreams of the depressed (1959), in which the presence of one or more specified elements was used to rate the dream as containing 'masochistic' content. This was also the rationale behind the/

/the SCE/PVE dichotomy of Molinari and Foulkes (1969); if any one or more of the elements dreamer talking, reflecting, or deciding were present then the whole dream was scored as containing secondary cognitive elaboration (SCE).

Ordinal rating scales are much more widely used. Hall and Van de Castle (1966) have provided a review of nominal and ordinal scales which have been used. A much more extensive review of virtually all scales used on dreams, which is in preparation for publication (Winget and Kramer, The Dimensions of Dream Content) lists many ordinal as well as nominal scales for measuring the occurrence of emotions, ego functioning, sexuality, orality, anality and hostility for instance. Such a wide variety of rating scales for the whole dream have been employed that someone searching the literature for the best one to employ could easily be overwhelmed. Many are merely 3, 4, 5 or often seven point scales rating a specified concept, such as bizarreness, or control, pleasantness, thematic coherence, plausibility, active control etc. One is faced with various choices: to invent one's own dimensions to suit one's own purposes, and so add to the plethora, is one way out. Another approach is to choose scales already used for specific reasons, of which the most obvious is that they have been used widely already since this facilitates comparison with other studies. The Foulkes' scale is one of the most widely used, and was designed for and has been "validated" on a number of studies of REM and NREM mentation. This scale was chosen for use in this study, for example, because it was predicted that the hypnotics should make REM dreams more like NREM mentation. (It is given in/

/in Appendix III). It is also unusual in that it combines several variables into one scale, and therefore carries a large amount of information which can be 'distilled' out by examining the occurrence of specific categories.

Without explicit theoretical expectations, however, and faced by a wealth of apparently partly overlapping dimensions, a more rational course of action can be adopted. Hauri et al (1967) chose 20 categories "conceptually discriminable but of possible overlap" and factor analysed them. They extracted 8 dimensions which between them accounted for about 60% of the total variance. These were vivid fantasy, active control, pleasantness, verbal aggression, physical aggression, heterosexuality, perception versus conception, and temporal reference. This system has been used successfully in at least one study since, that of Weiss and Foulkes (1970), a study comparing home and laboratory dreams. They found significantly ($p < .02$) more verbal aggression in home dreams, and trends for more physical aggression and more sexuality at home.

Content analysis on each element within a dream is clearly a much more laborious process. An early attempt to do this was the Hostility scale of Saul and Sheppard (1956), which they entitled "An attempt to quantify emotional forces using manifest dreams: a preliminary study". This and a similar scale for use in the analysis of (waking) verbal behaviour (Gottschalk et al, 1960) formed the basis for the series of scales devised by Whitman et al (1961). These scales were used in this study in an attempt to quantify latent hostility, anxiety and heterosexuality. They are/

/are given in Appendix II. In these scales, each phrase is scored on all the scales. The total of the scores for all the phrases is then calculated for each scale. Thus for each scale there is one score for the whole dream. These scales suffer the disadvantage that they are neither fish nor fowl: the scales themselves are ordinal, but the process of calculating a total score implicitly assumes they are interval scales. In comparing different dreams one is forced to rank order them.

There are two other ways in which these scales may be seen as ambiguous, though these aspects may not necessarily be seen as disadvantageous. The first is that the scales are allegedly for use on the manifest dream, but, as described in Chapter 13, they are clearly designed to be used as measures of both manifest and latent content simultaneously: the authors state that one must be aware of symbolic allusions etc. While this may be seen as a disadvantage, it is consistent with psychodynamic models of the dream which are not rigidly "Freudian". Freud himself (in The Interpretation of Dreams) made it quite clear that some dreams, or elements of them, could be read at a manifest level. While he applies this particularly to childrens' dreams, which are often simple undisguised wish-fulfilments, and while he regarded most adult dreams as necessitating analysis at the level of latent content, it is possible on a neo-Freudian or more general psychodynamic viewpoint to assume that the "meaning" of some parts of adult dreams can be read on a manifest level, and that some parts may require analysis of latent content. In this experiment/

/experiment, two of the rating scales (for sexiness and anxiety) were scored by a psychiatrist familiar with psycho-dynamic approaches. But certain common symbolic uses are widely recognised in everyday behaviour, and providing scoring is consistent it is possible to score some 'latent' content much as one would interpret jokes and allusions in everyday speech, where some references are taken to have both a literal and a symbolic intent. While it would be preferable to have separate scales for the latent and the manifest content, a scale need not be invalid because it includes both.

The other "ambiguity" in the scales of Whitman et al is of a similar nature. A single phrase can be scored on two or more scales for the same element. For example, if one character shoots another, this will be scored as 6 for (manifest) hostility, and also as 6 for (latent) sexuality, either homosexual or heterosexual as the case may be. Given the previous discussion, I think a justification for this can be made. Freud's concept of condensation signifies that more than one meaning can be attached to one element in the manifest dream. Given the (assumed) validity of such an assumption, it is clearly unexceptionable to score the same manifest element on two or more of the scales. Using the concepts of Hall's analysis (The Meaning of Dreams, 1966) if shooting someone can represent sex, then the choice of a gun as the symbol of sex (rather than, say, a flower) represents the dreamer's view of the sexual act as essentially aggressive. It is therefore logical to score such an element both on the sexuality and the hostility scales.

The Hall - Van de Castle system of content analysis:

Two major comprehensive systems of content analysis of the manifest dream have been produced in recent years. One is Foulkes and Shepherd's system for scoring childrens' dreams (1971). The other, previous, system is that of Hall and Van de Castle (1966). Some examples of this are given in condensed form in Appendix I. It has been used repeatedly, and was used in earlier form in three wellknown studies of Domhoff and Kamiya (1964 a,b,c).

The Hall - Van de Castle system deals solely with manifest content. It is a system of nominal scales. Some of them such as that for aggressive interactions could be treated as ordinal scales but the authors advise that they be used only at nominal level. There are scales for physical surroundings, characters, activities and social interactions, besides emotions, descriptive elements, success and failure and misfortune and good fortune. The discussion of the scales also includes norms derived from college students' home dreams (1000 in all), and a description of reliabilities.

The authors strict adherence to the manifest content of dreams, makes for what I would regard as one serious omission. Sexuality is covered by the scale for sexual interactions. As noted above, These are quite rare in comparison to the frequency of other interactions. Since their norms are derived from home dreams, and since laboratory dreams contain fewer sexual references than do home dreams, the frequency of sexual interactions is very small indeed in laboratory studies. (Domhoff and Kamiya, 1964a, quote only one fifth the/

/the number; Weisz and Foulkes, 1970, report ratings for sexuality in the laboratory roughly one third of that at home). This derives from the very constricted view of sex adopted by Hall and Van de Castle. Marriage they score solely as a friendly interaction (other activities can be scored on more than one scale). Sexual interactions are limited to those with physical sexual content, and fantasies of or propositions to physical sexual acts. Dating is regarded as non-sexual. Thus content with manifest sexual overtones ("she looked very attractive" - or even "she was looking very sexy") cannot be scored at all - on any of the scales provided. With this major proviso, their system is otherwise meticulously designed and presented, with excellent opportunities for high inter-laboratory reliability gained from following the stated criteria and practice with the examples given.

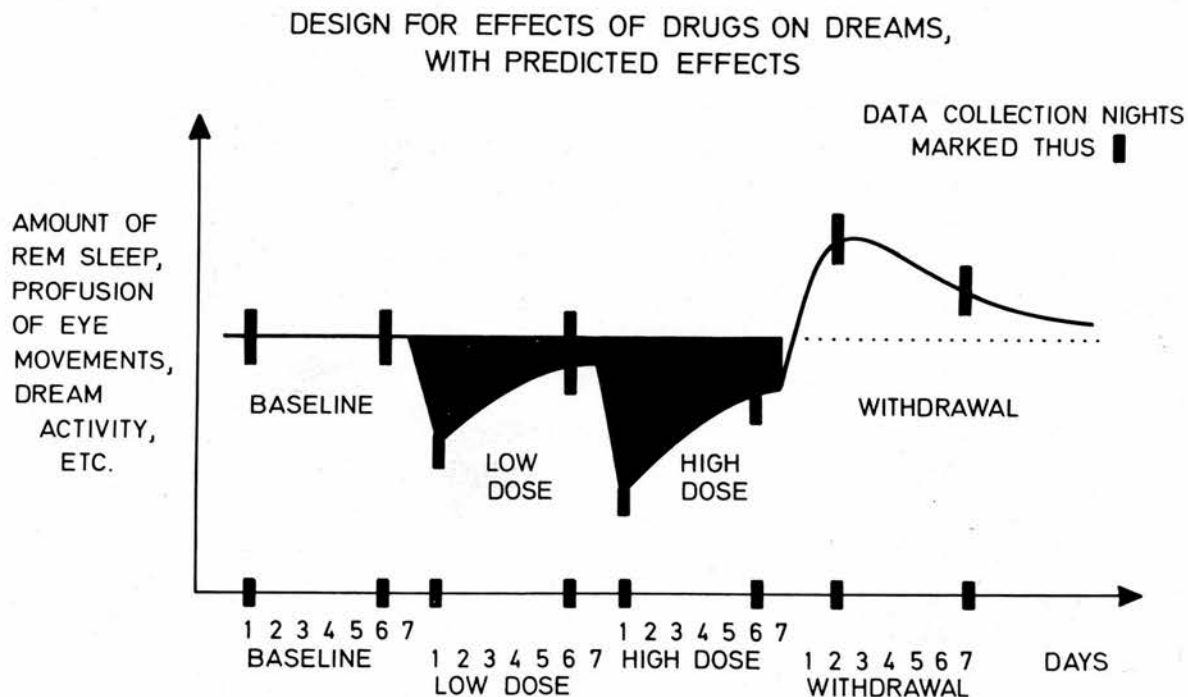


FIGURE 15. THE DESIGN FOR STUDYING THE EFFECTS OF DRUGS ON DREAMS.

On the drug it is predicted that the amount of REM sleep, eye movement profusion, and the 'intensity' of dreams will all be reduced. Tolerance will develop, and there will be a 'rebound' when the drug is replaced by placebo.

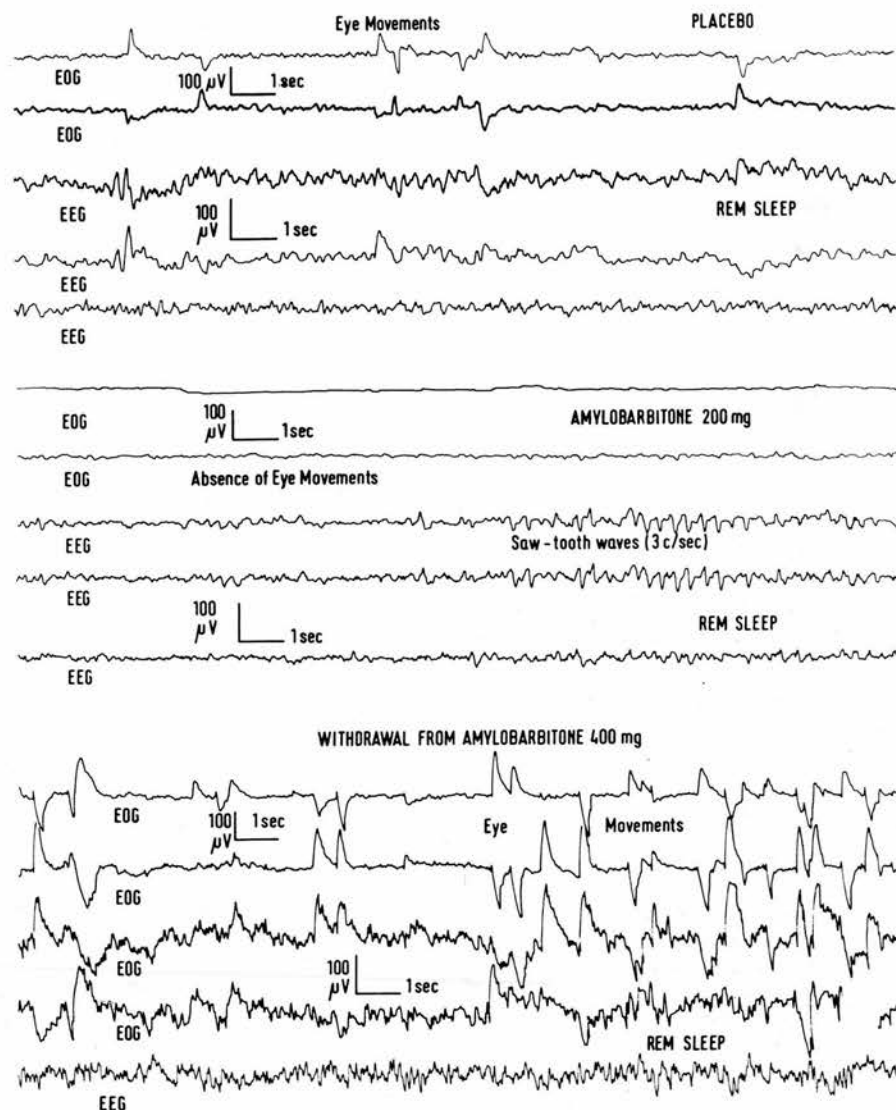


FIGURE 16. THE EFFECT OF AMYLOBARBITONE ON EYE MOVEMENT PROFUSION IN REM SLEEP.

Relative to baseline, eye movements are suppressed by the drug. On withdrawal, eye movements become very plentiful, and their amplitude is apparently enhanced. Saw-tooth waves in REM sleep are apparently not suppressed by the drug.

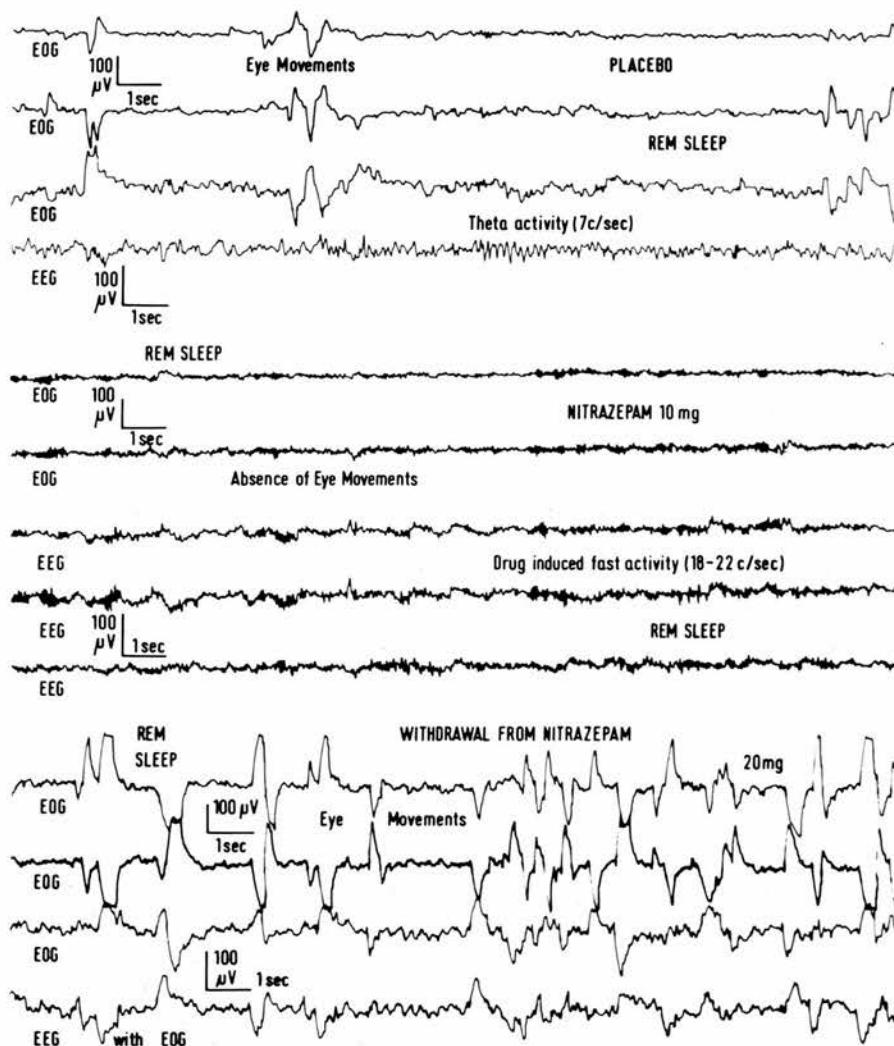


FIGURE 17. THE EFFECT OF NITRAZEPAM ON EYE MOVEMENT PROFUSION, AND ON THE EEG DURING REM SLEEP.

Nitrazepam suppresses eye movements in REM sleep. Its withdrawal leads to plentiful eye movements of high amplitude. Nitrazepam also produces striking changes in the EEG during REM sleep: fast activity becomes very prominent.

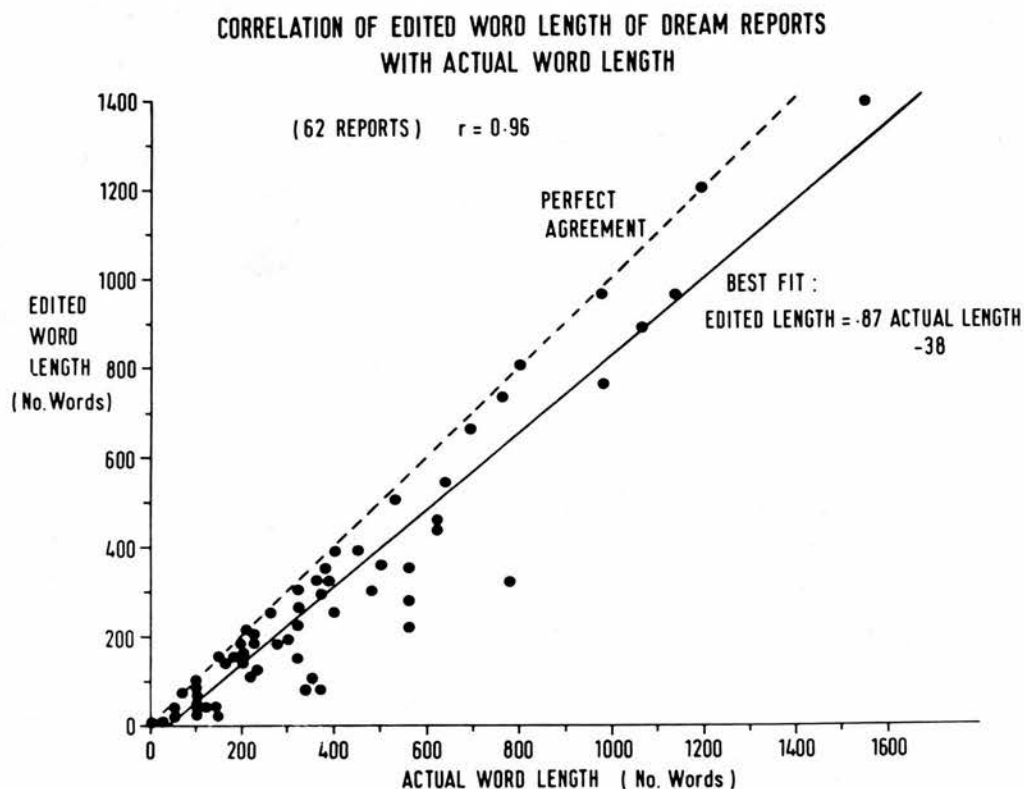


FIGURE 18. A COMPARISON OF THE LENGTH OF SUBJECTS' EDITED AND UNEDITED REPORTS.

Each dot represents one report. Two reports were so long they could not be included in the figure; they were however included in the calculation of the correlation.

RELIABILITY BETWEEN TWO JUDGES :

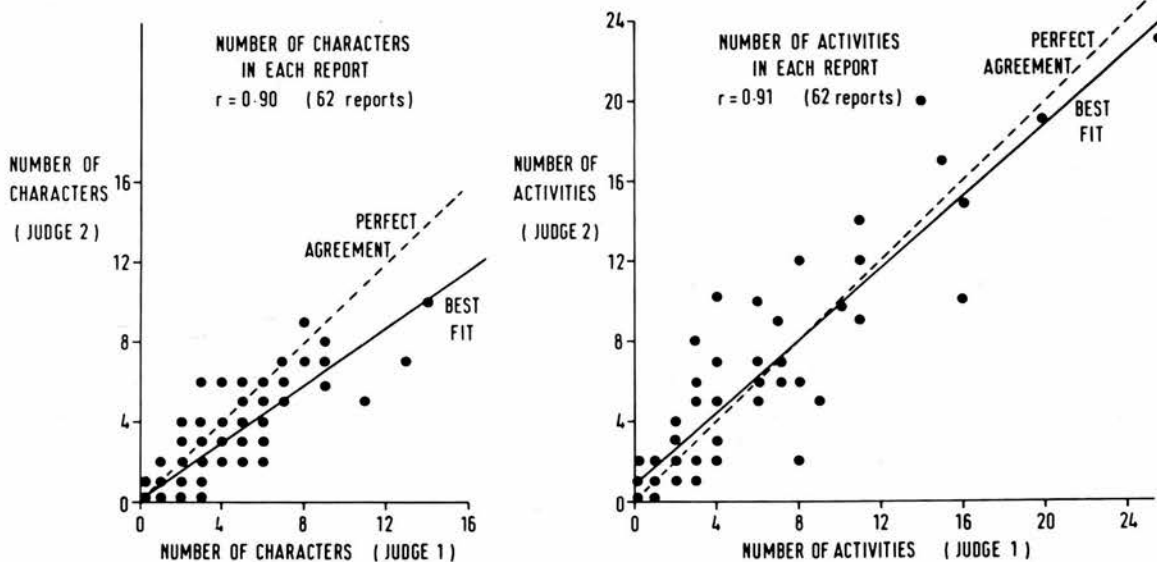


FIGURE 19. RELIABILITIES FOR THE HALL AND VAN DE CASTLE SYSTEM FOR SCORING THE NUMBER OF CHARACTERS AND THE NUMBER OF ACTIVITIES.

Each dot represents one or more reports.

TABLE 19.

SUMMARY OF THE DESIGN FOR WAKENINGS IN EXPERIMENT 1.Amylobarbitone group:

		Nights	1	6	1	6	1	6	2	7
			Baseline placebo	Drug Low Dose		Drug High Dose		Withdrawal placebo		
Subjects										
1	{REMP	2	+	+	+	+	+	+	+	+
	{REMP	4	+	+	+	+	+	+	+	+
2	{REMP	2	+	+	+	+	+	+	+	+
	{REMP	4	+	+	+	+	+	+	+	+
3	{REMP	2	+	+	+	+	+	+	+	+
	{REMP	4	+	+	+	+	+	+	+	+
4	{REMP	2	+	+	+	+	+	+	+	+
	{REMP	4	+	+	+	+	+	+	+	+

Nitrazepam group:Subjects

5

ditto

6

7

8

Placebo group:

9

10

ditto

11

12

TABLE 20.

SUMMARY OF THE DESIGN FOR WAEKNINGS IN EXPERIMENT 2.Amylobarbitone group:

Subjects	Nights	¹ Baseline placebo	¹ Drug low dose	¹ Drug high dose	² Withdrawal placebo
1	{ REMP 2 REMP 4	+	+	+	+
2		+	+	+	+
3					
4					
5					
6					
7					
8	{ REMP 2 REMP 4	+	+	+	+
		+	+	+	+

Placebo group:

Subjects	Nights	ditto	ditto	ditto	ditto
9	{ REMP 2 REMP 4	+	+	+	+
10		+	+	+	+
11					
12					
13					
14					
15					
16	{ REMP 2 REMP 4	+	+	+	+
		+	+	+	+

TABLE 21.

DREAMS

Please indicate whether you feel you dreamed a lot last night. A night which you feel contained an ordinary amount of dreaming should mean a mark in the centre.

- a) _____
- | | |
|---------------------------------------|---|
| Absolutely dreamless
in retrospect | Seemed to be vivid
dreaming all the time |
|---------------------------------------|---|

Could you now try and distinguish two elements: how much (or how long) you dreamed last night, and secondly, how vivid, bizarre or else everydayish, was what you dreamed about.

- b) _____
- | | |
|------------------------|--------------------------|
| Didn't dream
at all | Dreaming all
the time |
|------------------------|--------------------------|

- c) _____
- | | |
|--------------------------------|---------------------------|
| Very everydayish,
or boring | Very vivid, or
bizarre |
|--------------------------------|---------------------------|

ANXIETY

Please indicate, by a mark on the line, how calm or anxious you have felt mentally today. An average day should mean a mark in the centre.

- _____
- | | |
|--------------------|-------------------------------|
| Terrible agitation | Imperturbable
tranquillity |
|--------------------|-------------------------------|

TABLE 21(contd.)

CONCENTRATION

Please indicate, by a mark on the line, how well you have felt able to concentrate mentally today. An average day should mean a mark in the centre.

Extremely difficult
to concentrate

Wonderfully alert
and penetrating mind

TABLE 22.

NORMS FOR CONTENT ANALYSIS SCALES OF HALL AND VAN DE CASTLE

Norms from this study are derived only from those subjects who received placebo throughout the experiment. Wakenings resulting in reports with no content were excluded. To facilitate comparison with published norms, all figures have been converted either to percentage or to average numbers per dream.

	<u>This Study</u>		<u>Hall and Van de Castle¹</u> <u>(1966)</u>	
Number of dreamers (male)	8		100	
Number of dreams	61		500	
Word length	11 -	1193	50 -	300
	%	per dream	%	per dream
% dreams with any characters	95.1%		-	
Number of characters including dreamer		4.28		-
Number of characters excluding dreamer		3.48		2.36
% dreams with activities	90.2%		-	
Number of activities		4.68		4.72
% of dreams with aggressive interactions	25.4%		47.0%	
Number of aggressive interactions		0.37		0.80
% of dreams with friendly interactions	22.0%		38.2%	
Number of friendly interactions		0.41		0.50
% of dreams with sexual interactions	1.7%		11.6%	
Number of sexual interactions		0.02		0.15
% of dreams with any social interaction	39.3%		-	
Number of social interactions (total)		0.80		1.45
% of dreams with emotion	27.8%		-	
Number of emotions		0.49		0.56

¹Hall and Van de Castle's norms were collected from psychology students recording dreams as part of a class project. Hall and Van de Castle used 500 dreams from men and women, after they had excluded any dream less than 50 words long or more than 300 words long - an unnecessary and unfortunate criterion.

TABLE 23.

PUBLISHED RELIABILITIES OF THE HALL - VAN DE CASTLE SCALES

(These percentages are based on the total number of cases where either one or other judge scored the item as present).

	Sandler et al (1970) (Depressives) (Schizophrenics) (91 dreams) (217 dreams)	Hall and Van de Castle (1966) (Normal population) (100 dreams)
<u>Characters</u>		
<u>Presence</u>	83%	93%
Perfect agreement on number, sex, identify and age, if presence agreed.	75%	75%
<u>Activities</u>		
<u>Presence</u>	55%	-
Perfect agreement if presence agreed.	94%	85%
<u>Social Interactions</u>		
<u>Presence</u>	19%	-
Perfect agreement if presence agreed	68%	60%
<u>Emotions</u>		
<u>Presence</u>	50%	-
Perfect agreement if presence agreed.	88%	63%

TABLE 24.

INTER-RATER RELIABILITY OF SCORING ON HALL - VAN DE CASTLE
SCALES IN THIS STUDY.

	Pearson correlation coefficient (on number of eg characters in each report).	Agreement on presence <u>or</u> <u>absence</u> of any of the item in report, as per cent of <u>total</u> reports.	Agreement on presence of any of the item in report, as per cent of reports <u>where</u> <u>at least one judge</u> <u>scores item as present</u>
	N = 62	N = 188	N = 188
WORD LENGTH			
Edited/Unedited	.96	-	-
CHARACTERS			
Excluding the dreamer himself	.90	92%	90%
ACTIVITIES	.91	96%	96%
SOCIAL INTER- ACTIONS	-	86%	70%
EMOTIONS	-	92%	43%

Social interactions and emotions were both too scarce
to make calculation of correlation coefficients meaningful.

CHAPTER SIXTEEN

METHOD

1. Overall Design

The object of the experiment was to compare dreams 'on drug' with dreams 'off drug'. Such a comparison can be made two ways - by comparing the dreams of the same subject while on and while off the drug, or by comparing the dreams of two different groups of subjects, one of whom takes drug, the other acting as control group. The first sort of comparison is one frequently made in studies of the effects of drugs on sleep, especially since one of the chief objects of such studies is usually an examination of withdrawal effects. In such a design the subject takes placebo for a period at the start of the experiment, this period acting as a baseline from which subsequent drug effects can be assessed. Immediately following this 'baseline' period, the subject takes the drug for a period of time - say two weeks. Any tolerance effects which may develop should become evident during this period. The drug is then abruptly withdrawn (being replaced by placebo), and withdrawal effects are monitored for anything up to three months.

The second type of comparison is one more frequently used in experiments in the field of experimental psychology, where different groups of subjects are subjected to different treatments, and large numbers of subjects can be used. Such designs are also often used because there are easy methods of statistical treatment readily available for the analysis of results.

The bias of this author, and of a laboratory involved largely with drug studies, is towards the idea of using subjects as their own controls. There is, however, a distinct disadvantage of such a technique when applied to the collection of dreams. Dream content is very easily influenced by the laboratory situation (Domhoff and Kamiya, 1964a,b). It therefore seems possible that changes in dream content over the course of such an experiment might be due not so much to the drugs used, as to changes in the subject's relation to the experimental situation. This might be expected particularly during the 'withdrawal' phase, when at the end of the experiment the subject's dreams might be becoming more like 'home' dreams - more aggressive for instance (Weisz and Foulkes, 1970).

As a result of these considerations, it was decided to use both principles in the design of this experiment. Thus each subject underwent a baseline period (on placebo), a 'drug' period, and a 'withdrawal' period. Some subjects received active drug in the 'drug' period, while a control group received placebo in the 'drug' period - thus these subjects in fact received placebo throughout the experiment. Such designs, both using placebo control groups, have been recommended for general use by Lubin, 1971.

For the first experiment, therefore, it was decided to collect dreams from 4 subjects in each drug group: placebo, amylobarbitone, and nitrazepam. The experimental facilities allowed the collection of dreams from two subjects on any one night. It was hence possible to make part of the experiment double-blind. The experimenter (E)/

/(E) was unaware which of the two subjects were in the amylobarbitone and which in the placebo group. (This double-blind could not be maintained indefinitely since the experimenter had to read the subjects' EEG during the night, and the EEG is affected by the presence of barbiturates). The subjects on nitrazepam unfortunately had to be run separately, single-blind. Herman et al (1970) neatly showed the effect experimenter and subject bias can have on reports of mental life in sleep. They found dramatic effects with recall from NREM sleep, though the effect of bias was less, and was not significant, with REM awakenings.

The two subjects run each night were staggered as to their time of going to bed. This was necessary in order to avoid the possibility of E having to interview both simultaneously during the night. One subject went to bed at 10.30 p.m. and the second at between 11.30 and midnight. (Individuals were assigned early/late as far as possible to fit their normal bedtimes). Subjects were chosen for the barbiturate or placebo to ensure that half in each group went to bed early and half late. Since subjects in the nitrazepam group were run in pairs, necessarily half bedded early and half late. 'Early' and 'late' subjects were not distinguished at all in the analysis of the data - the division was only made to enable two subjects to be run concurrently.

The design so far involved a baseline period, a drug and a withdrawal period. For withdrawal effects to be noticeable it was reckoned that two weeks on the drug was essential. A dose of 400mg of amylobarbitone or 20mg nitrazepam (twice an effective hypnotic/

/hypnotic dose in each case) was regarded as one which would not be heavy enough to be disruptive of subjects' normal daily routines. To avoid giving subjects a large dose on their first night, it was decided to allow subjects one week on 200mg amylobarbitone (10mg nitrazepam) before receiving their first full dose. The design chosen was therefore one week of baseline (placebo), one week on low dose, one week on high dose, and one week following withdrawal. On the assumption that dream content is related to REM sleep and EMS, it was expected that maximal effects in the three 'treatment' weeks (drug, drug and withdrawal) would be on the first drug night, the first night on the full dose, and on either the second, third or fourth nights of withdrawal (Oswald and Priest, 1965; Evans et al 1968).

Experimental nights involved the experimenter staying up all night; therefore experimental nights could not feasibly be consecutive. It was thought suitable to collect data on two nights each week. One of these should obviously include the night when maximal effects were expected. The other night was chosen to be near the end of the drug week, to elucidate any tolerance effects. The nights thus chosen were: the 'first' and 'sixth' nights in the baseline week; the first and sixth nights on low dose of the drug; first and sixth nights on high dose; and the second and seventh nights of withdrawal. Such a design would, it was hoped, allow drug effects, dose effects, withdrawal effects, and tolerance effects to be examined for each of three groups: placebo, amylobarbitone and nitrazepam. The effects predicted are illustrated in Figure 15.

On each of these experimental nights, two awakenings were made. It was planned that these should be from the second and fourth REMPs of the night (subject to certain exceptions described in the procedure) - providing one awakening from the first, and one from the second half of the night. Awakenings were not made from every REMP in order not to deprive subjects of REM sleep during the night, by 'cutting short' their REMPs. Such deprivation taking place in the early part of the night could increase the "pressure" to REM and the "pressure" to dream later in the night: such an effect interacting with the other effects in the experiment could be a confounding influence, considering that an effect of this kind was found by Pivik and Foulkes (1966), using the Foulkes dreamlike fantasy scale.

The design as a whole is illustrated in Table 19 and in Fig. 15.

2. Statistical analysis

The design was constructed so as to allow for the use of analysis of variance on those measures meriting parametric tests. The analysis of variance (ANOVAR) used was a repeated measures analysis available on a computer program. The original program was written by M. Fearon, for the IBM 1620, in KINGSTRAN language. It was adapted by A. MacLean for the IBM 360/50, in FORTRAN.

For those measures derived for each awakening, three analyses were performed, one for each group of subjects (placebo, amylobarbitone, nitrazepam). Each ANOVAR extracted two main within-subject effects, namely REMP effects and NIGHT effects. The latter tested whether there were significant differences between any of the eight nights. If the ANOVAR showed that some of these differences were significant/

/significant, orthogonal comparisons (Edwards, 1968) were used to examine precisely which differences were significant. This procedure enabled determination of whether baseline/drug, drug/withdrawal, dose, tolerance, or any combination of these differences were significant.

This method of analysis was extended to other variables such as 'delay to sleep' - where there is only one measure per night - using 'NIGHTS' as the only within-subject main effect.

On two occasions each in the placebo and nitrazepam groups, subjects failed to have sufficient REMPs to allow two awakenings to be made in the night. Data for these cases was interpolated iteratively by the method of Cochran and Cox (1957 , p.110).

For these analyses of variance, separate error terms were used for the different sources of variance: Graham (1970) has pointed out the inaccuracies likely to arise from the use of pooled error terms. The error terms for each source of variance are illustrated below, for the basic analysis:

No:	Source:	MS tested against MS of Source No:.
1	Subjects	3
2	Nights	3
3	Subjects x Nights	
4	REMPs	5
5	Subjects x REMPs	
6	REMP x Night	7
7	Subjects x REMPs x Nights	

3. Subjects

Subjects were volunteers. They answered our largely word-of-mouth 'advertisement' that men aged between 18 and 30 would be paid roughly £1 a night for sleeping in the laboratory, and abstaining from alcohol and other drugs over a period of a month or so. In this experiment their age lay between 19 and 26. The only screening applied was an informal one by a psychiatrist to ensure that they were both mentally and physically fit to receive the drugs that were prescribed by him, and able to communicate mental experiences adequately for the experiment. One prospective subject was in fact dropped because he seemed unable to fulfil the second requirement and in the opinion of the psychiatrist was regarded as being too schizoid a personality. One other subject had to be replaced at the start of an experiment because he left unexpectedly after the adaptation-to-waking night.

Subjects were non-students, some known to the experimenter, most not known to him. They were all given a Maudsley Personality Inventory (Eysenck, 1959) and a questionnaire on work and sleep habits devised by the Research Unit on Intellectual Development of Edinburgh University. These revealed no significant differences between subjects assigned to the placebo group and those assigned to the amylobarbitone group on either the E or N scales of the MPI (t test), nor on any of the 20 questions in the questionnaire (chi-square). One of the questions in the questionnaire asked subjects how often they normally recalled dreams. Roth et al (1972) have recently shown that volunteers and non-volunteers for experiments on sleep/

/sleep and dreams differ in a surprising way: volunteers are persons who recall fewer dreams spontaneously at home than the non-volunteers. It is therefore worth noting that subjects assigned to the various drug groups did not differ on this variable.

The mean scores on the E and N scales of the MPI for all 20 Ss in both experiments were less than one standard deviation from the population means given by Eysenck (1959).

4. Adaptation nights

It is well known that one's first night's sleep in a strange environment is rarely as 'good' as a night at home. This effect has been documented, and the 'first night effect' as it is known to sleep-researchers, may last for more than one night, though by the third night most parameters have reached stable values (Schmidt and Kaelbling, 1971). The effect consists of an increased delay to sleep and increased arousals during the night - and also of disturbances to the 'internal' structure of sleep - a reduction in the amount, and proportion, of the night spent in REM sleep, and even a reduction in the proportion of REM sleep actually containing eye movements (Rechtschaffen and Verdine, 1964 ; Agnew et al 1966 ; Mendels and Hawkins, 1967 ; Schmidt and Kaelbling, 1971).

Because this effect extends to the profusion of eye movements within REM sleep, and since this measure is affected for at least two nights, it was thought important to give all subjects adequate adaptation to the laboratory procedures. They were all given two nights in the laboratory with full electrode fixings, to allow/

/allow them to adapt to the laboratory per se. A few subjects who found it especially difficult to adapt to the laboratory situation in that they took an abnormal time to go to sleep - two hours, for instance - were given an extra night at this stage. All subjects were then also given one night to adapt to the waking procedures to be described later. This was done so that subjects could become accustomed to the idea of being woken up in the middle of the night and asked what was going on in their mind; and so that they should find it less difficult to go back to sleep after being woken up on the experimental nights. On this night (which I shall refer to in future simply as 'the adaptation night') data was collected as far as possible exactly as on any experimental night. Because problems were liable to arise on this first waking night - e.g. the subject might not understand the questions being put to him - the data was not regarded as being up to the standard of other nights, and were excluded from the bulk of the analysis proper, although the 'dream' reports were scored like any other so that an inspection could be made for any obvious 'adaptation' effects. The data from these nights were not however included in any of the statistical analysis. To do so would have been a very dubious procedure since the adaptation night contained a very large number of 'no content' reports compared with the other nights (see Fig. 34). There were also occasions when awakenings were not made according to the scheme: for instance, one subject took so long to go back to sleep after his first awakening that he had had no further REM sleep before breakfast time.

In order to accustom him to the waking procedure and interview, therefore, he was woken from NREM sleep and interviewed shortly before he was due to get up for breakfast.

5. Procedure

Each night that the subjects came into the laboratory they were given tablets to last them for that night and until their next night in the laboratory - the pills being placebo, active drug, or a mixture as the case might be. Each night's tablets were packed in a separate, dated envelope. On experimental nights subjects took their tablets immediately before retiring to bed, and they were instructed to do the same on the nights at home. Subjects were forbidden to take any drug for the duration of the experiment, except nicotine, tea or coffee, which they were allowed to take freely. They were also allowed to take aspirin in moderate doses if required.

As mentioned earlier, two subjects were run each night, one going to bed at 10.30 p.m. and one between 11.30 p.m. and midnight. EEG, EOG and EMG recordings were made with the electrode placements described in Part A. (Skin potential and heart rate were not recorded in these experiments). The recordings were made on a Beckman type TC 12-channel electroencephalograph. The subjects slept in separate but adjoining rooms.

Subjects were normally woken from their second and fourth REMPs. However, because it was important to control for time-of-night as much as REMP number, certain exceptions were made. (There is no study known to me which has attempted specifically to parcel out the effects of REMP and time of night on dream content). It sometimes happens that subjects 'miss' their first REMP - the latency to the first REMP being of the order of 150 mins instead of in the more usual range of 50-100 mins. In these cases it was thought appropriate to take/

/take the first and third REMPs instead. A criterion for this was fixed: if the delay between sleep onset and the start of the first REMP was greater than 135 minutes, then the first and third REMPs were used.

A second exception to this rule occurred when REMPs lasted less than 7 minutes. In these cases the next REMP was used instead. Thus it sometimes happened (chiefly on active drug nights) that the second and third REMPs might be used, for instance if a delay to REM sleep of 140 minutes eventuated in a REMP of only 5 minutes duration.

As a check on whether these controls operated effectively, the time-of-waking was analysed with all the other variables, in the hope it would show no significant effects - besides the REMP effect, since there must obviously be differences between the times of the second and fourth REMP!

Four subjects were run in what turned out to be a pilot study. These subjects were woken after 15 minutes of REM sleep, unless the subject went into stage 2 after $7\frac{1}{2}$ minutes, in which case he was woken immediately. This procedure resulted in subjects on the active drug being woken after significantly less REM sleep than those on placebo - because on active drugs, not only is total amount of REM sleep reduced, but also REMPs tend to be broken up by fragments of NREM sleep. With the length of REM sleep preceding waking not controlled between the groups, it was clearly necessary that the awakening criteria should be altered.

Because on such hypnotics as were used REMPs do not always/

/always last for as long as 15 minutes, it was decided after the pilot experiment to give subjects $7\frac{1}{2}$ minutes of REM sleep before being woken. If they entered stage 2 or stage 1 or 0 after 7 minutes of REM they were immediately woken and interviewed, but they were not woken after less than 7 minutes REM. Because the REMPs, even of normal subjects on placebo nights, are frequently broken up by short periods of NREM sleep, it was necessary to have some criteria of how much stage 2 sleep interspersed in the REMP would be tolerable. Wolpert and Trosman showed in an early study (1958) that 'dream' recall falls off rapidly in the few minutes following the end of a period of REM sleep. It was arbitrarily decided that snatches of NREM sleep lasting 2 minutes or less in among a REMP would be tolerable. If, however, NREM sleep lasted for more than 2 minutes, only to be followed by a return to REM sleep, then the REM before the interruption would not be included in the calculated 7 minutes. The 7 minutes was calculated on the basis of the actual number of epochs scored on the spot as REM sleep.

To illustrate how this procedure operated, two examples are given below:

- 1) REM -- 5 min -- /NREM 1 min / REM $2\frac{1}{2}$ min/ wakening.
- 2) REM -- 2 min - / NREM -- 6 min - - - /REM -- $7\frac{1}{2}$ min - / Wakening.

Both waking and sleeping EEGs are affected by the presence of drugs in the brain, and as a consequence (particularly with the higher doses) the discrimination of REM from NREM sleep can become quite difficult. With the virtual absence of eye movements at the full/

/full doses this problem becomes particularly acute. All EEG records were effectively scored twice for that part of the night near the awakening time: once on-the-spot (and by necessity often in haste) and once later on. Since there were sometimes differences between these two scorings, the actual times spent in REM sleep prior to awakening were subjected to a 'control' analysis of variance to ensure that subjects in all conditions were in fact getting the same amount of REM sleep. These times were of course those derived from the later, considered, scoring of the EEG records.

Two other 'control' variables were measured (but were only included in the experiment part way through). These were the latency from the onset of the wakening stimulus to the subject being awake (as defined from the EEG record), and the latency from the stimulus to the subject's first word of content, in reply to E's first question. It was hoped this way to ensure that any differences between placebo and drug nights would not be attributable to the Ss taking longer to 'wake up' on the drug. Goodenough et al (1965a), for instance, have shown that 'thinking' reports are more liable to result from gradual awakenings than from abrupt awakenings.

One other variable recorded was the time Ss took to go back to sleep - or more precisely, the total time they remained awake, from the wakening stimulus until the first stage 2 sleep¹.

¹Note that time awake here is comparable to the way the term is used at the start of the night: "Sleep latency" also treats stage 1 as wakefulness rather than sleep, whereas during the bulk of the night it is conventional to treat stage 1 as being sleep rather than wakefulness.

The Interview: At the end of their $7\frac{1}{2}$ minutes of REM sleep, the subjects were awakened over an intercom system by 'feedback' -- something between a low whistle and a screech, depending on the volume. (A moderate volume was used initially: as the nights progressed subjects generally woke more readily, so less volume was required; occasionally however, volume had to be increased because a subject failed to wake readily). The feedback was given in a short burst, this being repeated if necessary. When the subject was judged awake, he was addressed by name. When he answered the interview was begun. This procedure was demonstrated on the evening of the first waking night.

The interview opened with the question "What was passing through your mind just before I signalled?". This question was designed not to bias the subject as to whether he had been dreaming, or even to suggest to him that he was necessarily asleep when his thoughts were interrupted by E. If the subject could not recall anything (or failed to answer at all for one minute) he was prompted with "Can you recall anything at all?". After their spontaneous reports, subjects were asked "Anything else?", "Anything before this?", and "Any more details?", until they replied in the negative to each question. This form of questioning was preferred to a more directive interview firstly because it greatly reduced the possibility of bias in the way E can ask his questions. Secondly, after a 'prompting' type of interview was tried (of the form "can you tell me any more about ...?") with four pilot subjects mentioned earlier it was found that more script was obtained than could be dealt with readily. Some/

/Some subjects in this situation tended to repeat essentially the same information over and over again. There are only two ways of avoiding bias in questioning: to ask no leading questions or to exhaust all possible ones. While further questioning, if it is pressed, will often reveal a certain amount of extra information, it is impracticable with some subjects to continue asking questions till they dry up: some of them will quite happily talk all night! Given this, it seems better to lose a little information and remain neutral in questioning. It must be remembered that after seeing the EEG in REM sleep the experimenter often could not avoid guessing which subject was on placebo and which on active drug.

In order to ensure that vital information was not lost through the subject being unaware it was wanted, after the interview described above a set of direct questions was asked. These questions were modified in the light of experience, but were identical for any group of four subjects throughout all their experimental nights.

The four placebo and four amylobarbitone subjects were asked:

"Were you awake or asleep when I signalled?"

"Would you say you were thinking or dreaming?".

"Were you observing or participating?".

Since all subjects developed an obvious 'set' that they had been asleep when signalled to, the subjects who received nitrazepam were not asked that first question. They were asked the other two questions, and in addition two others:

"Were there any people?" OR

"Have you mentioned all the people?".

"Did you have any feeling or emotion, pleasant or unpleasant, associated with this?".

After this part of the interview, subjects were asked whether there was anything else they could recall, anything else they had not mentioned, and when they finally answered 'No' to these whether they were "Sure?". At this point they were thanked and told they could go back to sleep. Many apologised when they could recall nothing; it was insisted to them that this didn't matter: they were thanked all the same.

Subjective estimates: These subjective estimates consisted of ten centimetre lines (illustrated in Table 21) for anxiety, concentration and dreams. The subjects filled these out daily. Ten-centimetre lines are visual analogue scales which are convenient for self-rating. Their advantages have been described by Aitken (1969) and Zealley and Aitken (1969). It was postulated that anxiety should be reduced by both sedatives, and would increase to above baseline values on withdrawal. Concentration was monitored as a routine check that subjects were not suffering the effects of the drugs enough to cause subjective impairment of their normal life. For the first experiment, only the first of the 'dreaming' lines was used. The other two were used only in the second experiment in an attempt to discriminate subjects' perception of how 'intense' their dreams were from how long they spent dreaming. Subjects filled in the lines every evening (anxiety or concentration) or morning (dreams), whether or not they attended the laboratory that night. The score extracted from the lines is simply the distance (in cm) of the subject's mark from the left or right hand side of the line.

6. Analysis of physiological data

The EEG records were scored for the whole night using 20 sec epochs according to the standard criteria of Rechtschaffen and Kales (1968), except for the departures mentioned in Chapter 5. The measures utilised were: delay to sleep onset at the start of the night, delay from sleep onset to the first REM sleep in the night, time awake following each wakening, and the actual number of minutes of REM sleep preceding each wakening.

The two variables latency to sleep, and latency to REM sleep, were examined not as an integral part of the experiment, but as additional information. The sleep latency might give an idea of whether these sleeping pills were performing their function. However, since they were taken immediately before retiring it was not expected that they would necessarily take effect fast enough to affect sleep latency. Also, sleeping pills may not be able to hasten the onset of sleep in healthy young subjects who don't take long to get to sleep anyway.

Since the subjects were being woken in the night, it was obviously not meaningful to calculate the proportion of the night spent in REM sleep and compare this with normative values. One other index is available, however, of the effect of hypnotics on REM sleep: the latency to the first REM of the night (Oswald and Priest, 1965 ; Oswald, 1968). This index was used because latency to the first REM is clearly unaffected by the wakings later in the night.

In addition to this, a measure of the frequency of the eye/

/eye movements themselves was attempted. The counting of actual numbers of eye movements has been used in order to "quantify" REM sleep in the past (Lewis, 1968b) but the counting of every single eye movement is an exceedingly laborious task. A modification which has been used before is to divide the normal 20 or 30 epochs used for scoring sleep stages into smaller epochs, and count the number of such epochs which actually contain one or more eye movements. The size of epochs chosen here was 2 seconds. For each REMF from which an awakening was made, the number of 2 sec epochs with eye movements (EMs) in the 5 minutes of REM sleep preceding the wakening was counted. Although it would in theory have been an advantage to do this scoring blind, this was not in practice possible, because the EEG during a REMF of a heavily sedated subject is distinguishable immediately from that of a subject on placebo (see Figures 16 and 17).

This measure of the 'profusion' of EMs was made to verify that the drugs used which it was predicted would reduce the profusion of EMs, were in fact doing so. This effect was tested using the analysis of variance described already. For this purpose, on those occasions when only one wakening was made in a night (because there only 3 REMFs), instead of interpolating the missing values iteratively as for the other variables, a count of 2 sec epochs was made in the last REMF not actually used for a wakening.

Eye movement profusion scores were also calculated to test directly the hypothesis that 'intensity' of dream content is related to 'intensity' of the physiological activity of the REMF. For each individual REMF for which a wakening was actually made, a point biserial correlation/

/correlation was calculated (Guilford, 1965) between the EM profusion measure and the active/passive ratings described later in this chapter.

A word is in order here about the statistical treatment of these measures. Measures such as the delay to sleep, or the delay to REM sleep cannot be assumed to be normally distributed. The delay to REM sleep may be bi-modally distributed, with peaks at around 60 and 150 minutes. This measure was therefore subject to non-parametric analysis of variance (Siegel, 1956). Variables such as the delay to sleep are unimodally distributed, but there is generally marked skewness in the distribution. Scheffe (1959 p.337) has pointed out that the effect of deviations from normality is slight on inferences about means. Nevertheless, logarithmic transformations were applied in experiment 1 to all variables suspected of being markedly skewed. The resulting analyses of variance were then compared with analyses on the untransformed figures. Transformation resulted in more conservative F ratios for sleep latency, the profusion of eye movements, and also for the verbal report length (skewness in the latter distribution is evident in Figure 18). Transformation had no effect on F ratios for any of the measures of dream content (number of characters, etc). Hence logarithmic transformations were applied to sleep latency, EM profusion and report length before all analyses. Scheffe also discussed the effects of unequal variances upon inferences about means. Provided cell numbers are equal (as they are throughout these experiments), the effects of unequal variances are not serious (Scheffe, 1959 ; p.345,353). Variance ratios as high as 10:1 will only increase the probability of type 1/

/1 errors from .05 to .06. A check was made anyway on variance ratios for sleep latency and BM profusion. On active drug the variance of these measures could be reduced along with their mean values. Comparing drug and withdrawal values, this was indeed true. After logarithmic transformation, however, in only one instance was there a significant ratio as judged by the F test (Guildford, 1965); for nitrazepam, the ratio of variances of sleep latency was 2.57 between drug and withdrawal conditions. It should be clear from the discussion above that with equal cell sizes, such a variance ratio will have a negligible effect on the validity of inferences from ANOVARS.

7. Analysis of the dreams

All the reports recorded in the night were later transcribed, typed and coded. These reports were the raw data for the 'dream' content analysis. The first and most obvious measure to be derived from these reports was their length. The number of words ("word-length") was counted: this included all words, excepting only those sections of the report which contained no information. Such sections were answers to the questions in the structured part of the interview which offered no new information, and answers to any question which consisted of mere negatives (e.g. "No, I thought I could, but I can't recall any more"). It would have been possible to use various other definitions of word length designed to include only the 'meat' of the content - not counting articles, interjections, repetitions etc. - but this was deliberately avoided. It is common to regard/

/regard repetitions especially as somehow 'redundant' information in the report. It is my view that on the contrary they may contain information about what the dreamer regards as the important parts of the dream. Particularly since one of the content scales used (Whitman et al 1971) demands the scoring of repetitions, it was felt important to include everything which could conceivably be regarded as content.

Three different approaches to the evaluation of content were used. These can be grouped into whole-dream ratings, 'content analysis' (which attempts to detail the manifest contents of reports in a replicable fashion), and a scoring system based on psychodynamic assumptions about the significance of the dream report.

Some of the whole-dream ratings, described shortly, were made by a judge blind as to the conditions from which the reports had been collected. Otherwise all scoring and rating of content was done by myself. In addition, an experienced scorer from another laboratory (John Trinder) scored all the dreams in experiment 1 on the Hall and Van de Castle scales; he was blind both as to the waking conditions and as to which reports came from which subjects. The scoring done by myself was clearly not 'blind', since I had collected the reports. However, all reports were identified by a code number, and by the time I came to score the reports, I could recollect the waking condition of only a very few. Reports from any one subject were all scored together. The first 18 reports scored were scored again at the end. No notable differences in scoring occurred.

(a) The content analysis/

(a) The content analysis: Hall and Van de Castle's system (1966) for the content analysis of dreams is probably the most comprehensive. It was used here in the expectation that it would provide a detailed picture of the changes in dream content under the influence of the sleeping pills. It tells us not only how many different characters appear in the dream, but whether they are friends or enemies, young or old etc. Therefore it was anticipated that it would provide the most comprehensive picture of changes in dream content available.

The scales chosen for use were those for Characters, Social interactions, Activities, Success and failure ("achievement outcomes"), Good fortune and misfortune ("Environmental press"), and Emotions. Summaries of these scales are given in Appendix I. These not used were the scales for physical surroundings (Objects, and Settings) and the 'Theoretical scales' of Castration wish, Castration anxiety, Oral incorporation, Penis envy, Oral emphasis and Regression.

Two modifications, or additions, were made to the Hall and Van de Castle scale for Characters. The first was that the dreamer himself was scored as a character ('D') if he appeared in the dream events. Hall and Van de Castle decided to omit the dreamer "since (he) is such a constant factor in almost every dream". However, they do include the dreamer in their scales for interactions and activities - it only seemed consistent therefore to include him in the Characters scale. It appeared particularly necessary since in this study it was thought likely that one of the chief differences between drug and placebo dreams would be that placebo dreams would include the dreamer as/

/as an active participant while drug dreams might not include him at all. The dreamer was scored as present only if the subject said he was present, or if the dreamer was involved in some activity - he was not scored as present if the subject said merely 'I was observing'.

The second addition to the character scales concerned whether a character was actually present or not. In the Hall and Van de Castle system, a character is scored when he is mentioned in the report, whether or not he actually appears (e.g. "The police were supposed to come"). Hall and Van de Castle do, however, give five different and distinct conditions for the scoring of characters (1966, p53): physical presence, being heard or seen but not physically present, mentioned but not present (example above), referred to in relation to another character ('I went into my brother's room), and the appearance of a part of the character ('I held my boyfriend's hand). I scored characters 'present' if either the first or the last of these conditions were fulfilled; otherwise the character was scored as not physically present.

The content analysis system of Hall and Van de Castle is based upon categories. They are at pains to avoid the idea that murder for instance, is any 'more' aggressive than being rude to someone. Each act, character, emotion is scored into one category. The end product therefore is a sort of shorthand list of all the events of the dream: one young male, one strange girl, one murder, one theft, one offer of help, and so on.

For the purposes of this experiment, a lot of this information would be expected to be redundant. Although when the reports were scored, all information was tabulated for each scale, it was never/

/never intended to use all of it. There seemed no reason for instance, to suppose that the sleeping pills would have any effect upon the age of the characters in the dreams. While the scales themselves are nominal in type (murder/theft/destruction are separate aggressive categories), the initial interest of this study was in questions like "Were there fewer aggressive interactions on amylobarbitone?". For this reason most of the categories of the scales were ignored; the data used in the statistical analysis were the number of characters, the number of characters of the opposite sex, the number of aggressive interactions, and so on.

The measures subjected to the analysis of variance were:

Total number of characters including dreamer.

Total number of characters excluding the dreamer.

Number of characters actually present.

Number of unfamiliar characters¹.

Number of male characters.

Number of single characters, (as opposed to groups of two or more).

and

Total number of activities, and the number of activities with the dreamer as the initiator.

The frequencies of social interactions, emotions¹, success/

¹ Characters are divided into 'familiar' and 'unfamiliar' according to Hall and Van de Castle's division (1966, p.163). Unfamiliar characters are those identified as strange, those identified only by their occupation, or by their ethnic origin, and characters of uncertain identity. Others are scored as familiar: members of the family, relatives, those known to the dreamer, and 'prominent people'.

/success, failure, good fortune and misfortune were all so low as to justify only an analysis by chi-square of the number of reports with emotion, etc. (see the norms in Table 22). Analyses using chi-square were also made of the number of reports with specifically aggressive interactions, with specifically friendly and sexual interactions, the number with interactions initiated by the dreamer, the number of reports in which the dreamer himself appeared (with or without others), and finally the number of reports in which there were no characters or just the dreamer on his own. REMP effects on these measures were tested by the sign test (Siegel, 1956).

Corrections for the word-length of a report have been advocated: Hall and Vande Castle recommend their use (1966, p.13). Trinder et al (1970) even produced a regression formula to correct for the effect of dream report length on measures such as the number of characters. It was decided in this study however not to use such corrections unless there was a significant effect of the experimental conditions on word length. For the purposes of this study, if a significant effect of a drug on, say, the number of activities can be demonstrated, it is felt that this should be treated as a 'real' finding. If a significant effect on word-length were also to be found, then a correction could be applied and a further analysis made of the drug effect on activities-per-word. But no need is seen for the application of such corrections, without a clear theoretical reason for doing so. A subject economical in his use of words will naturally require more words to describe a dream with many activities. His reports may/

/may show a very high correlation between report length and the number of activities. If some effect reduced the number of activities in his dreams, he would need less words to describe it. It would clearly be absurd in this situation to "correct" the number of activities in the dream for the report length. The existence of an association between two variables is not in itself a ground for removing the effect of the first on the second.

(b) The psychodynamically oriented scales: Some of the scales described by Whitman et al (1961) were chosen. These scales had recently proved capable of discriminating changes in dream content during the menstrual cycle in two young women studied in our laboratory (Burns and Lewis, unpublished). More importantly though they were chosen because they allowed the scoring of symbolic references, in contrast to the Hall and Van de Castle scales which restrict themselves rigidly to the scoring of manifest content. This is especially evident in Hall and Van de Castle's inclusion of sexual content - they consider only physical sexual interactions, or fantasies of them, and ignore any subtler sexual interaction. Since it was clearly predicted that sexual dreams would increase in frequency on withdrawal of active drugs (as in Carroll et al, 1969 and Lewis and Oswald, 1969), this was an important consideration.

The scales actually used were the Hostility¹, Anxiety and Heterosexuality scales from Whitman et al (1961). These scales/

¹Largely because Whitman et al only sketch in the use of their scales, recourse was made for the Hostility scale to its origin in Saul and Sheppard (1956), who provide full instructions to enable the scale to be used with confidence.

/scales are given in Appendix II. The Dependency scale was not regarded as relevant to the hypothesis under test. The Homosexuality, Motility and Intimacy scales were tried out with one pair of subjects. Their use was not continued because they were found exceedingly difficult to score: Whitman et al do not offer good guidelines and examples in the way Hall and Van de Castle do.

The scales Whitman et al constructed are implicitly interval scales, although in their paper Whitman et al used non-parametric statistics to test their findings. Each phrase receives a score of between 0 and 6, and these scores are added up to provide a total raw score for the dream. It is in fact quite clear that the scale is not an interval scale, in so much as, for instance, a death threat cannot be considered three times as hostile as the threat of discomfort. For this reason the procedure employed by Whitman et al themselves was employed: the raw scores for each report were ranked and a Friedman non-parametric two-way analysis of variance was then performed (Siegel, 1956). In order to do this it was first necessary to average the scores for the two REMPs on each night: the two factors for the analysis were thus Nights and Subjects. The effect of REMP was tested separately by a sign test (Siegel, 1956).

Since, unlike the Hall and Van de Castle system, these scales score each repeated mention of some interaction as if it were a fresh interaction, an analysis was also performed on the raw scores divided by word length. Thus high scores could not solely be attributed to/

/to a verbose subject who repeats himself. It will be noted that on the Hall and Van de Castle system, a verbose subject who repeats himself will not score any higher than a subject not prone to repetition.

(c) The Rating Scales: Since this study originated from the finding that 'active' dreams tended to come from REMPs which were more active in terms of the number of eye movements, and since one of the principal findings of Carroll et al (1969) was that barbiturates made more dreams 'passive', the dreams were rated as 'active' or 'passive'. The criteria were those of Berger and Oswald (1962): dreams were rated 'active'.... "according to the nature of the events described, and especially if (the judge) felt such events would have been accompanied by many shifts of gaze, had they occurred in real life". The same judge (I.O.) was used to rate the reports as in the 1962 study. It was therefore possible to make the scoring of the reports in this part of the study totally 'blind': I.O. had never seen the EEG or been present at the time of recording. He was told which dreams came from a given subject (subjects were labelled A,B,C), and what proportion of the reports were collected under the 'drug' nights - but he did not know which subjects received active drug and which received placebo throughout.

While scoring the reports in this way, this independent but experienced judge also tried to guess whether each report came from a placebo, drug or withdrawal night.

Finally, he rated the reports for sexiness, for anxiety, and for psychotic thinking. Each report was rated as either 0, 1 or 2.

These ratings were based on clinical feelings about the dream content.

The judge described how he rated the dreams thus:

Sexiness: "A clinical dream interpretation, at a superficial level, of the symbolism, etc. e.g. "I was climbing stairs and found my mother waiting for me at the top and saying I'd been a long time coming" is scored as 2. "

Anxiety: "Again a clinical interpretation of the described dream situation, not just when the dreamer reports 'panic' etc."

Psychotic thinking: "A measure of 'psychoticness' or dreamlike/bizarre/colourful/emotional/perceptual/adventurous quality".

All the reports were also scored on Foulkes' dreamlike fantasy scale (Foulkes, Spear and Symonds, 1966) (see Appendix III). This scoring was not designed to be used with an unstructured, non-directive interview; however, it was in practice easy to apply to the extended information given by most subjects. When this experiment was started it was felt more important to maintain the non-directive interview than to mould it to the requirements of any one scale. Moreover, the Hall and Van de Castle system of content analysis was initially regarded as central to the experiment. It was thought worth while also using Foulkes' scale despite the fact that it was not intended to be used without the structured interview, since Carroll et al had reported effects of barbiturates using this scale. In the second experiment, a compromise was reached in that all the necessary questions were included in the structured part of the interview at the end.

Chi-square tests were used to examine the effects of the experimental variables on the Foulkes' rating scores. Individual tests were performed for the several 'factors' of which the 8-point dreamlike fantasy scale is made up: recall/no recall of specific content; Conceptual/perceptual; real/not real, at the time; and everydayish or bizarre.

Chi-square tests were not used, however, for the comparisons between REMPs within one condition (night) - sign tests were used for testing REMP effects with all the rating scales.

(8) Reliability of the dream content scales used:

(a) Published reliabilities for the Hall and Van de Castle system:

Hall and Van de Castle (1966) devote a chapter to the reliability of scoring. They discuss the different ways of describing reliabilities of dream scoring: percentage of agreement, and coefficients of correlation. These two measures can be used for different purposes: in particular, measures of percentage agreement can be used to express a variety of things, depending on how they are defined. If characters in a dream are scored by four symbols, for number (one, or a group), sex, age and identity, then one measure of agreement between judges is the 'percentage of perfect agreement' which examines the number of times judges agree on the scoring of every symbol for every character in the dream report. Other percentage agreement scores can be meaningfully used, however. For instance it might be suitable to look at the number of characters, or the number of male characters, scored by the judges for each report. These measures of reliability will give different values for 'percentage agreement' figures.

The measure chosen must clearly relate to the purpose for which the measure of reliability is required. Even the widely used correlation coefficient can mask differences in scoring if, for example, one judge always scores twice as high as another judge.

For the Character scale, Hall and Van de Castle quote the following percentage agreements between two judges (these are based on 100 dreams);

Presence of a character :	93%
Number (single/group) :	92%
Sex :	89%
Identity :	81%
Age:	92%
Perfect agreement :	76%

(These are percentages of all occasions where one or other judge scored a character).

For the Activities scale, where perfect agreement involved agreement about the type of activity and also about the character(s) involved, they quote a percentage of perfect agreement of 85%. For Social Interactions, judges must agree on scoring for both the characters involved, the nature of the interaction, and whether it was initiated, mutual, reciprocated or self directed: the percentage of perfect agreement falls therefore to 54% for aggressive interactions, 61% for friendly, and 64% for sexual interactions. However, agreement on all but one item is reached on 70-72% of the occasions where one or other judge scored an interaction. For Emotions, Hall and Van de Castle quote agreement between two judges on the class of emotion in 63%.

/63% of cases where one of them scored an emotion.

These reliabilities were all derived from the two authors of the scales separately scoring one set of dreams. Both of these 'judges' were therefore not only experienced at scoring dreams, but importantly, they had co-operated in setting up the scales they were scoring. Necessarily, they therefore both 'understood' the scales, and shared the same assumptions as to how the various categories were to be interpreted. What happens to these reliabilities when the two scorers came from different laboratories? Sandler et al (1970) had two raters score sets of dreams without prior consultation. There were in fact two sets of dreams scored, one from depressives, the other from schizophrenics. Sandler et al reported somewhat lower rates of agreement than Hall and Van de Castle for characters, little difference for activities and social interactions, and, surprisingly, a higher reliability for emotions (see Table 23). One of the points mentioned by Sandler et al was that the disagreements between the two judges were often consistent - they reflected clearly distinct interpretations of the scoring rules. Sandler et al comment that if the two judges were to have consulted in order to iron out these differences the reliabilities could have been considerably improved.

(b) Reliabilities for the Hall and Van de Castle system in this study

All the dreams in this experiment were in fact sent to John Trinder of the Veterans Administrations Hospital, Cincinnati, who scored them all ('blind') on most of the scales of the Hall and Van de Castle system. This was done so that the dream reports could be scored by a judge/

/judge with experience in using the system as well as by this author (who was at that time inexperienced in scoring dreams!). This also enabled a check on the reliability of scoring to be made. We scored the dreams independently and quite without any prior consultation. The other judge first edited the dreams of all repetitious material and of E's questions, so that the reports read as coherent passages. The word length was counted from these edited reports. Figure 18 shows the relation between the word length of edited and unedited reports. These edited reports were scored on the Characters, Activities, Social interactions, Emotions, Success and Failure, Misfortune and good fortune, Descriptive elements (Modifiers, Negatives and the Temporal scale), and Physical surroundings scales.

Two ways of looking at the reliability of the scoring have been employed. One is the calculation of reliabilities analogous to those described above, for those scales on which both this author and the other judges scored the reports. These results are presented in Table 24, and in Figure 19. Because the interest in this experiment was in the numbers of various types of items in a given report, rather than upon the precise identification of individual characters, interactions, and so on, the reliabilities were calculated on how many characters (for instance) there were in each report. Because Hall and Van de Castle's correlation coefficients are based on the number of items scored in groups of five reports, their coefficients are not comparable and I have not troubled to quote them. For the rarer events (Emotions, Social Interactions), Table 24 gives the number of dreams in which the judges/

/judges agreed an event took place.

The other indication of 'reliability' came in the analyses of variance. The scores of the second judge for the number of Characters, number of Activities, number of Negatives and report length (words) were all submitted to the same ANOVAR as were this author's scores. A comparison was thus made whether differences in the judges' scoring had any material effect on the results of the experiment. This is particularly interesting in view of two important differences between the judges. One has already been discussed: the fact that one judge edited the dream report while the other avoided any editing. The other is that in calculating the number of activities: this judge counted a 'mutual' activity as one activity; the second judge counted such activities (and social interactions) as two activities. Out of nine analyses of variance two differed as to significant effects. Both of these were disagreements as to whether between-subject differences were significant.

(c) Reliabilities of the other scales: Whitman et al (1961) merely state that on their seven scales their "interjudge reliability was at the level of .75"; they do not describe on what this reliability was calculated. However, Saul et al (1954) in their description of their Hostility scale (the same scale as was published in their 1956 paper, which was used in this study, and formed the basis for Whitman's scale) quote rank difference correlations among the scores of three judges as .85, .84 and .83.

The inter-rater reliabilities quoted by Foulkes for his/

/his dreamlike fantasy scale were based on studies which used his interview format. From his own laboratory, he quotes a Pearson r of .97 for the reliability of individual report classification, on 150 reports from 20 subjects (Pivik and Foulkes, 1968). Carroll et al (1969) reported a reliability of .94 between two judges previously unfamiliar with the scale who rated reports collected using the standard interview. A second judge (Stuart Lewis) rated two dozen of the dreams in this study (two chosen randomly from each of twelve subjects). Spearman's rank correlation coefficient between Lewis' ratings and my own was .96.

Carroll et al also report an inter-rater reliability for the 'active-passive' rating originally used by Berger and Oswald (1962). The inter-rater reliability of .90 was between two judges, one of whom (I.O.) was the judge in the 1962 report and also in this study. Stuart Lewis also rated the two dozen dream reports mentioned above as active or passive. His agreement with the other rater (I.O.) was 75%. The contingency coefficient (Siegel, 1956) between the two sets of ratings was .69, which is significant at the .05 level. This would appear to be a much lower reliability than that found in the Carroll et al study, but it is not clear from their report whether the two reliabilities were calculated so as to be comparable. They merely say " r " was 0.90.

Reliabilities for the ratings for sexiness, anxiety and psychotic thinking are not available; the only time one of these has been used before was in the Carroll et al study, when the rating for sexiness/

/sexiness was used, but only one of the two raters in that study employed it.

9. Summary of predictions

It was predicted that, compared with the control group receiving placebo throughout, the effects of both amylobarbitone and nitrazepam would be to:

Reduce the time taken for subjects to fall asleep;

Increase the latency between sleep onset and the first REMP;

Reduce the time taken to get back to sleep after awakenings;

Reduce the profusion of eye movements in REMPs;

Reduce the number of characters, activities, social interactions and emotions, in the 'dream' reports;

Reduce the rated anxiety, hostility, sexuality and psychotic thinking in the reports;

Decrease the proportion of 'active' reports in favour of 'passive' reports;

Make reports from REMP interruptions more like NREM mentation reports - specifically to make mental activity conceptual rather than perceptual, everydayish rather than bizarre, and less real-at-the-time;

On the 'intensity' hypothesis, one of the independent judges (John Trinder) also predicted that the drugs would reduce the 'dramatic intensity index' - a global index of 'intensity', derived by him from the total number of social interactions, emotions, successes, failures, misfortunes and good fortunes in the report/

/report. (Activities are curiously not included in this list.)

On the 'quality' hypothesis, it was predicted that the sedatives would make dreams more pleasant (and withdrawal make them more noxious). On this hypothesis, therefore, it was predicted that:

The number of negatives in the report would be reduced by the drugs;

The number of aggressive interactions would be reduced, and the number of friendly and sexual interactions increased relative to the total number of interactions;

Similarly the number of 'negative' emotions, failures, misfortunes, and negative evaluation adjectives (on the descriptive elements scale) and the number of unfamiliar characters would all be decreased by the active drugs in favour of their 'positive' equivalents.

The effects of withdrawal were expected to be the reverse of the changes described above.

10. The design of the second experiment

When the results of the experiment just described had been analysed, they appeared somewhat inconclusive in a couple of respects. In particular, the effects of the drugs on the proportion of active and passive dreams was on the margin of significance. In order to be able to resolve this uncertainty and describe the results with confidence, it was decided to run the experiment on more subjects. For this/

/this experiment, only one of the two drugs was used, namely amylobarbitone, again with a placebo control group in a double-blind experiment.

Experiment 2 was designed so that data from the first experiment could be combined with the data from the second. Because no effects of dose, or of tolerance between nights one and six of drug administration had been found, it was not thought necessary to concentrate on these variables. It was therefore possible to have dream collection nights once instead of twice a week (the strain of night work was telling on the experimenter!).

Data were thus collected from night one of a baseline week, a low dose week, a high dose week, and night two of withdrawal. In order to keep conditions for these subjects as similar as possible to those the initial subjects had undergone, the procedure for the subjects was identical to that in experiment one, excepting that on night six of each week, subjects slept undisturbed in the laboratory instead of being woken up. It was felt particularly important to keep the subjects coming in to sleep in the laboratory as often as in the first experiment, so that laboratory influences on both the actual sleep (first night effect) and on the dreams would be as similar as possible in the two groups.

Four subjects were again run in each group, again in a double-blind procedure, with one subject on amylobarbitone and one on placebo for each pair of subjects - one going to bed early and one late.

The results from these subjects were combined with the data/

/data on the comparable nights from the subjects in the first experiment. Thus in the analysis there were eight subjects in the amylobarbitone group and eight in the placebo control group, each recorded for four nights. This is illustrated in Table 20. Adaptation nights for the subjects in this experiment were identical to those for subjects in the first experiment.

The interview format in the second experiment: Because the results of the first experiment showed that Foulkes' dreamlike fantasy scale was one of the more useful scales, the interview format was altered in the second experiment so as to include all the questions used in Foulkes' own standardised interview.

In Foulkes' description of how to use his scale, he says that although most of the rating is done by the subject himself, one element in the rating should be done by E, not by the subject: whether the dream was "everydayish" or "bizarre". Nevertheless, I asked subjects themselves whether they thought their dream was everydayish or bizarre, although I sometimes chose to ignore the subject's answer. This was done in a few cases where subjects treated events taking place in the laboratory as ipso facto bizarre rather than everydayish, whereas to the experimenter references to the laboratory are instances of the subject's dreams being concerned with mundane, immediate, 'work', rather than with the bizarre or fanciful.

The questions put to the subject after the initial non-directive questioning were thus as follows:

"Was there any visual imagery: did you see anything?"

"Did it seem real at the time, or were you aware at the time that you were thinking or dreaming?".

"Were you observing only, or were you participating?".

"Would you describe the content as everydayish or bizarre?".

"Have you mentioned all the people? OR "Were there any people" and

"Did you have any feeling or emotion, pleasant or unpleasant, associated with this?".

The dream content ratings used in the second experiment:

Because the object of the second experiment was to confirm the doubtful findings from the first groups of subjects, the dream reports in this experiment were not scored on all the systems used in the first experiment. They ^{were} rated on the Foulkes' dreamlike fantasy scale, as active or passive, and for sexiness, anxiety, and psychotic thinking. The judge who performed the active/passive ratings, and rated the reports for sexiness, anxiety and psychotic thinking, once again attempted to guess whether the reports came from placebo, drug or withdrawal nights.

The psychodynamically oriented scales of Whitman et al were not used in this part of the study since they had yielded not even a suggestion that they were able to discriminate placebo reports from reports on active drug.

On the content analysis scales of Hall and Van de Castle, the scales for Characters, Activities, Social interactions and Emotions/

Emotions were used; those for Success and failure and for Misfortune and good fortune were dropped as having proved of no value.

One innovation was tried. I had hoped that having become familiar with both placebo and 'drug' dream reports, there were certain cues I had learned which would enable me successfully to guess which reports came from which conditions. Such a procedure could not of course be truly blind, since although the reports were now coded, I had been present at their collection. The attempt was in fact a total failure: even though I had been present when all of them had been collected, I was unable to guess (let alone remember!) their origin any better than chance.

CHAPTER SEVENTEEN

RESULTS

1. In brief:

It had been hypothesised that the two drugs, nitrazepam and amylobarbitone, would reduce the profusion of eye movements in REM sleep, and associated with this, they would make content from REM awakenings "thought-like" rather than "dreamlike", less "real", less bizarre and more everydayish, "passive" rather than "active". It had been expected that reports from subjects on active drugs would contain fewer characters, fewer activities, fewer social interactions and less emotion than placebo reports. And it had been expected that drug withdrawal would lead to sexy, active, bizarre dreams full of people and probably emotional.

The actual results were a sad disappointment in comparison to these confident expectations. The judges were no better than, and even worse than chance in discriminating placebo, drug and withdrawal reports. There was no relation between the proportion of active or passive dreams and the drugs. Foulkes' dreamlike fantasy scale, which has been successfully used in discriminating REM from NREM sleep onset reports (Foulkes, Spear and Symonds, 1966) and was apparently capable of discriminating barbiturate from placebo reports in the study by Carroll et al (1969), proved unable to discriminate barbiturate from placebo reports in this large study. The scales of Whitman et al did not reveal any increased sexuality, hostility or anxiety on drug withdrawal - neither did Oswald's rating scales for sexiness, anxiety and psychotic thinking. The results of analyses of the Hall and Van de/

/de Castle system of content analysis proved almost entirely negative.

The only positive effects of the drugs were that, first, nitrazepam had an effect in making dreams everydayish, and its withdrawal made them bizarre. Secondly, nitrazepam reduced the number of dreams with looking and watching activities on the part of the dreamer. Thirdly, there was a significant effect of amylobarbitone on the number of characters in the reports, when data from all eight subjects were used.

Certainly these are meagre effects in comparison with what had been predicted. More so since the effect of the two drugs on the profusion of the eye movements themselves was quite dramatic: full doses of both the drugs nearly abolished the eye movements.

There was a relation between eye movement profusion and whether dreams were active or passive, but though significant, it was slight. Since it was only visible when all reports from all twenty subjects were pooled, and was not visible in terms of a parallel effect of the drugs on EMs and content, it would seem that the association between EM profusion and dream content is a tenuous one indeed.

2. Physiological measures:

(a) Sleep latency, latency to first REM, and REM length:

Nitrazepam had a significant effect upon sleep latency (Table 25). This effect was not pronounced, however, and was only evident in that withdrawal produced difficulty for the subjects in getting to sleep: they took roughly twice as long to go to sleep on the withdrawal/

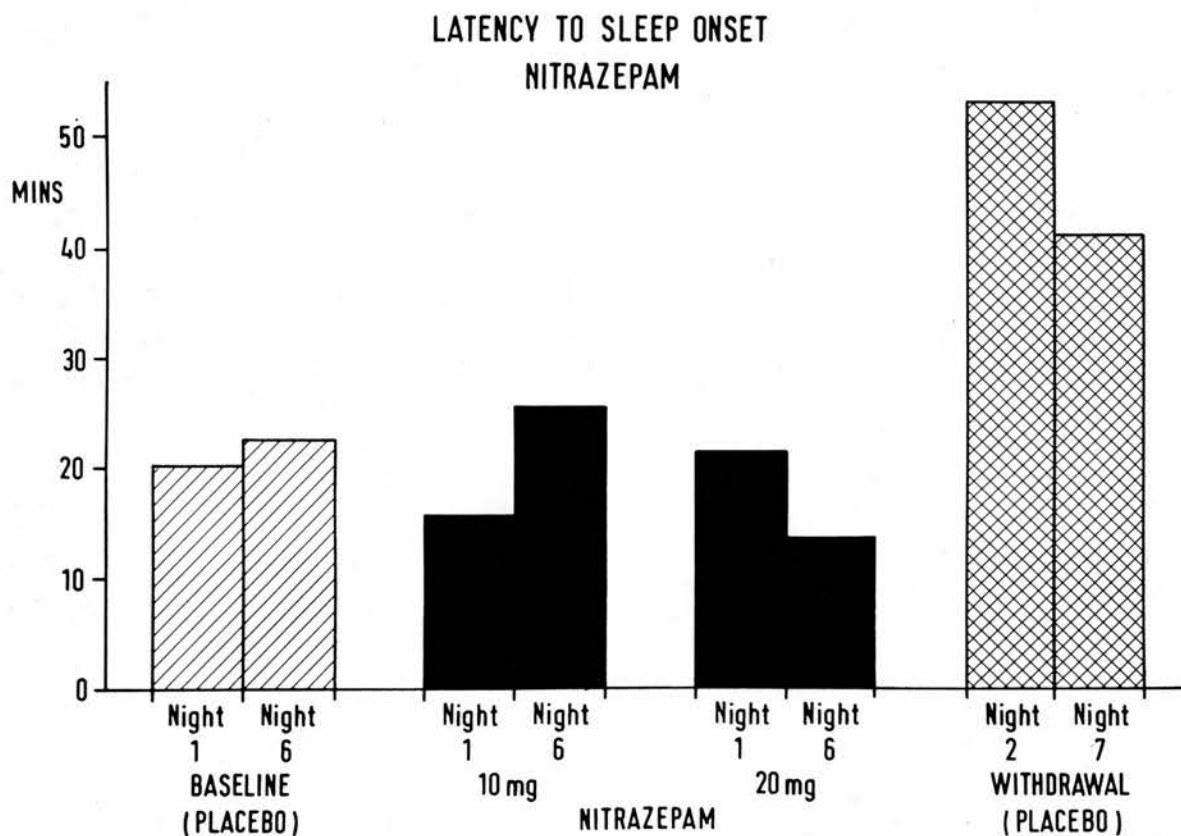
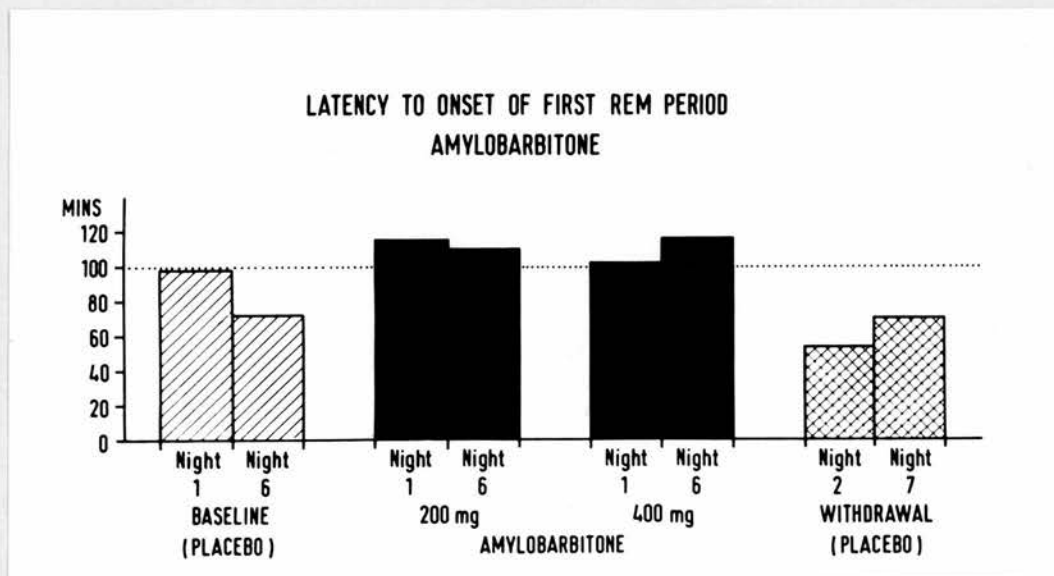
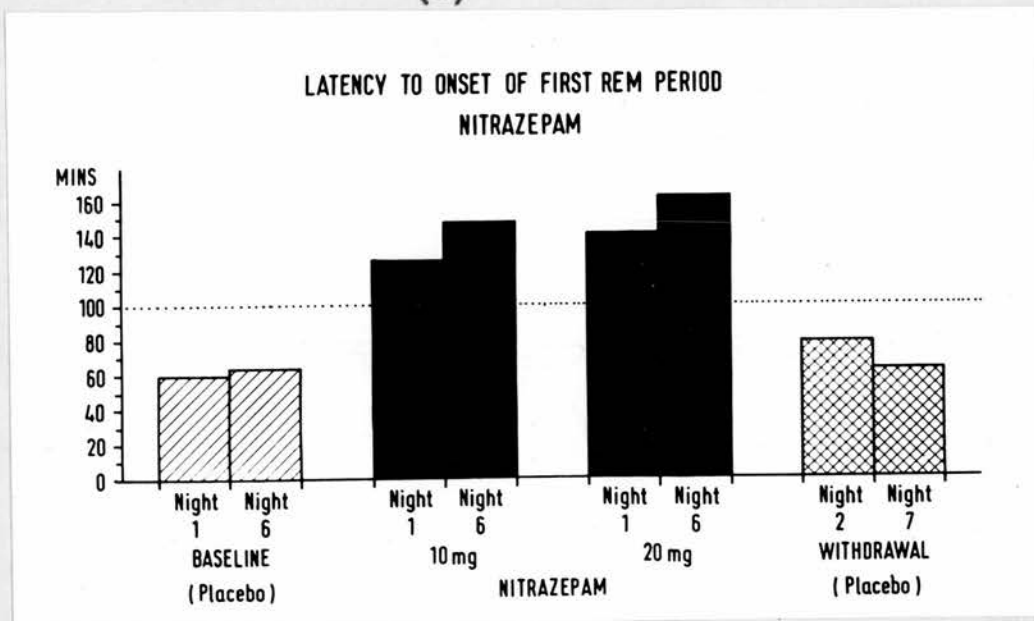


FIGURE 20. LATENCY TO SLEEP ONSET. EFFECT OF NITRAZEPAM.

It took subjects significantly longer to fall asleep following the withdrawal of nitrazepam. This effect is still striking one week after withdrawal.



(a)



(b)

FIGURE 21. LATENCY BETWEEN THE ONSET OF SLEEP AND THE FIRST REM.

(a) The effect of amylobarbitone

(b) The effect of nitrazepam

Results are shown in each case for four subjects in Experiment 1. Note that the mean latency is less than 100 minutes on all placebo nights, and greater than 100 minutes on all active drug nights. This effect is significant with nitrazepam, where the first REM occurs roughly 90 minutes later on the active drug.

EFFECT OF BARBITURATE ON SLEEP DISTURBED BY DREAM COLLECTION

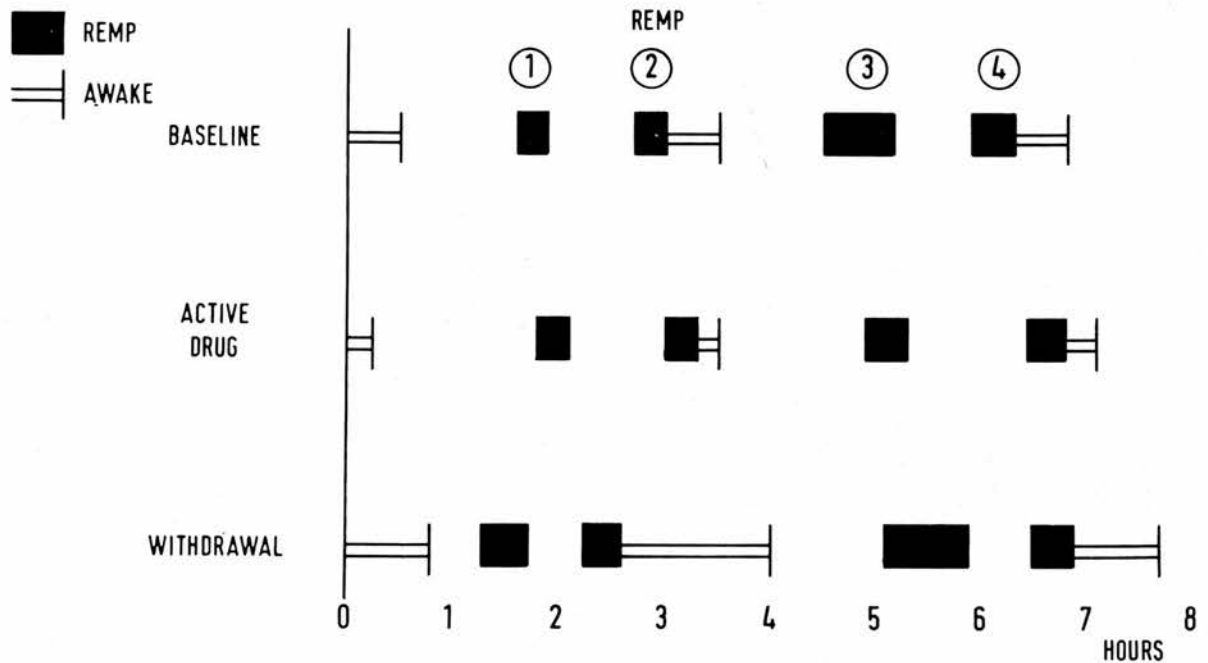


FIGURE 22. EFFECTS OF AMYLOBARBITONE ON SLEEP PATTERNS IN THIS EXPERIMENT.

The diagram attempts to show the interaction of two effects. One is the action of amylobarbitone in delaying the onset of REM sleep. The other is the effect of the drug on the time subjects spend awake after they have reported their 'dreams'. This latter effect is demonstrated in the next figure (Fig. 23).

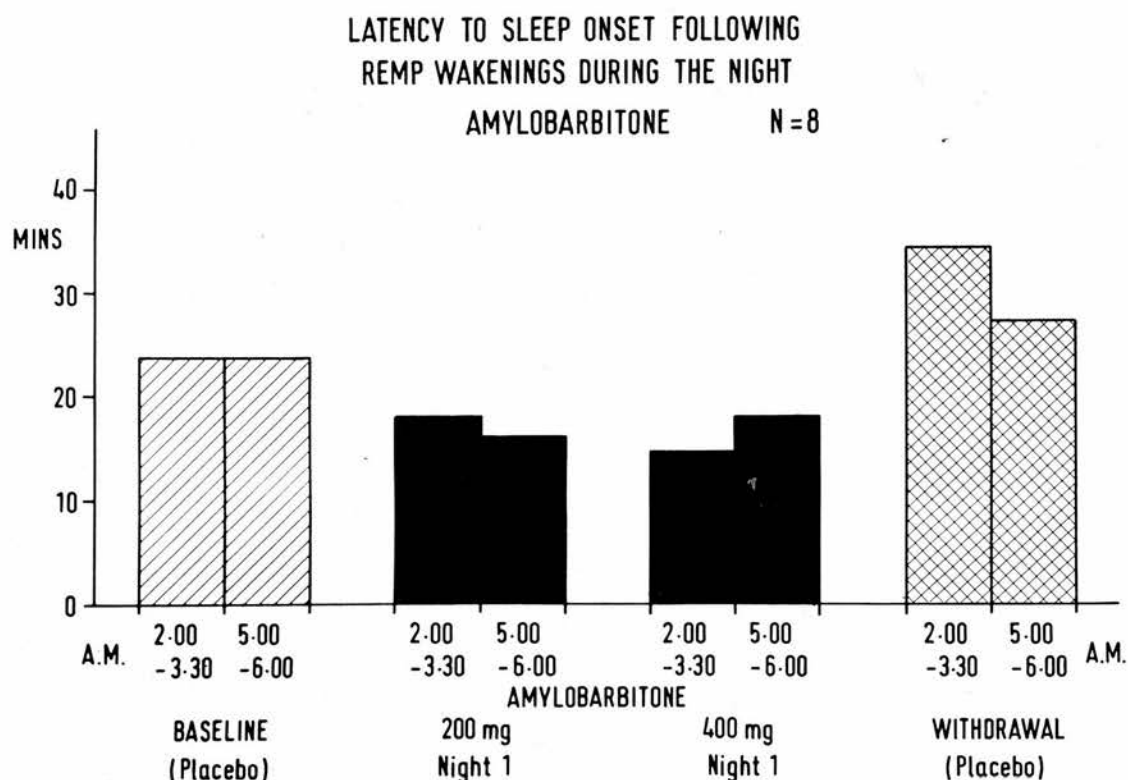


FIGURE 23. THE EFFECT OF AMYLOBARBITONE ON THE TIME SUBJECTS SPEND AWAKE IN THE NIGHT AFTER THEY HAVE BEEN WOKEN FOR A 'DREAM' REPORT.

Results are presented for the early (approximately 2.00 a.m. to 3.30 a.m.) and the late wakings in each condition. Results are from both experiments 1 and 2. This time includes the time spent actually telling the 'dreams'.

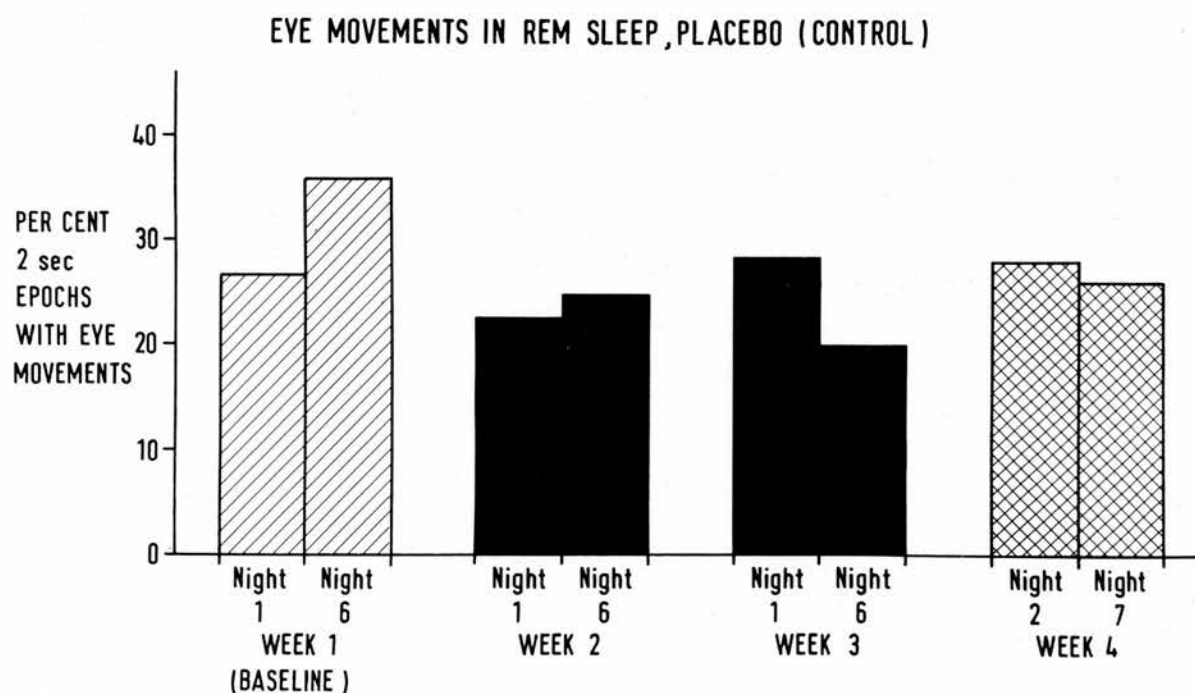


FIGURE 24. EYE MOVEMENT PROFUSION OVER FOUR WEEKS OF THE EXPERIMENT IN SUBJECTS RECEIVING ONLY PLACEBO.

Data from Experiment 1.

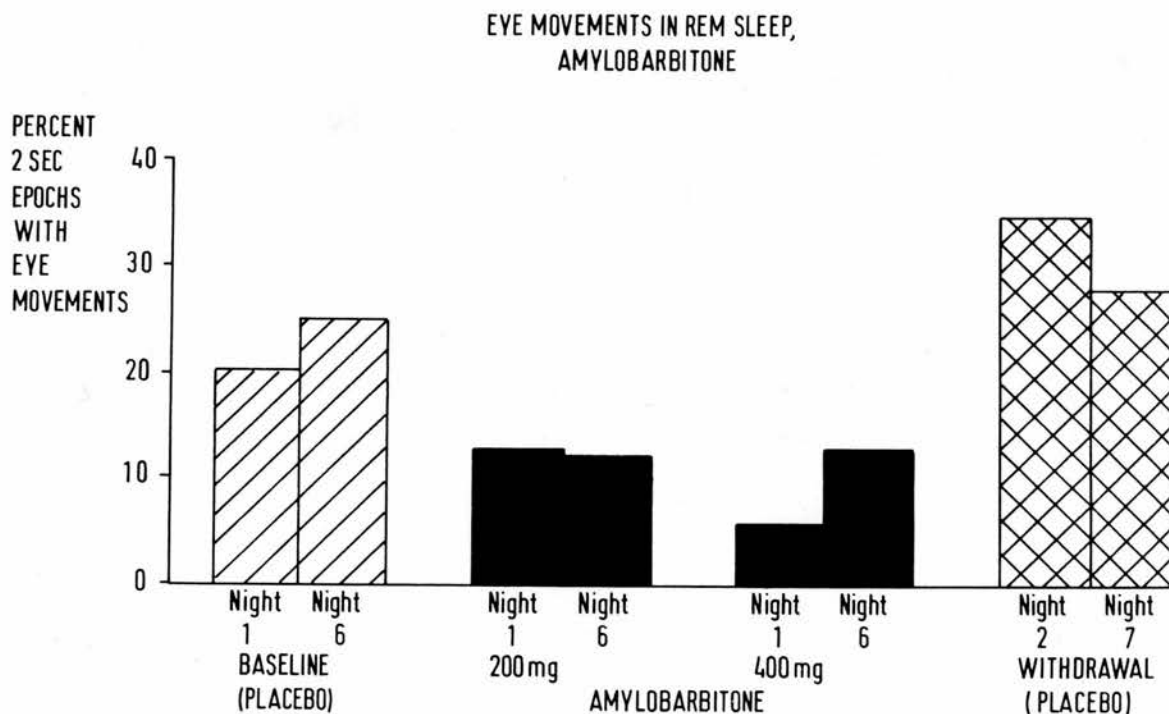


FIGURE 25. EYE MOVEMENT PROPUSION: EFFECT OF AMYLOBARBITONE.

The effect of the drug is highly significant. Dose effects, tolerance effects and withdrawal effects are not significant.

Data from Experiment 1. The results of Experiment 2 were essentially similar.

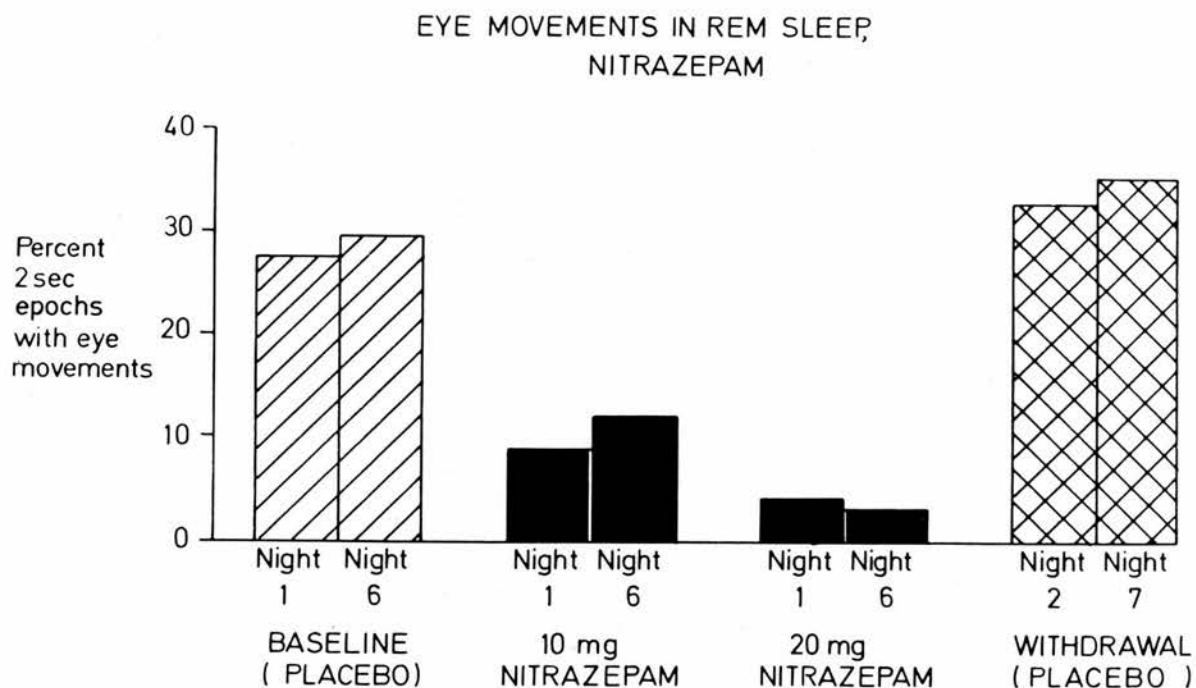
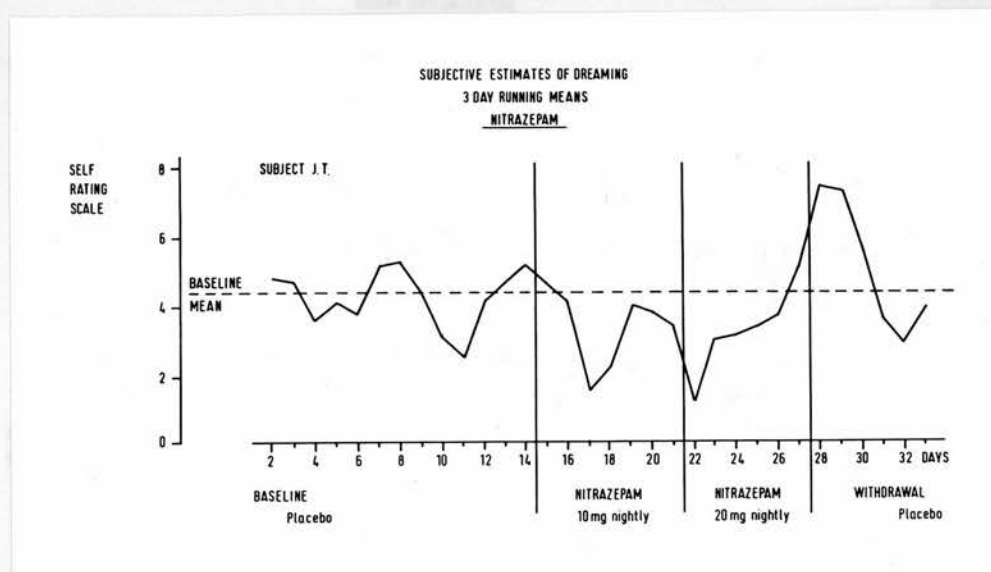
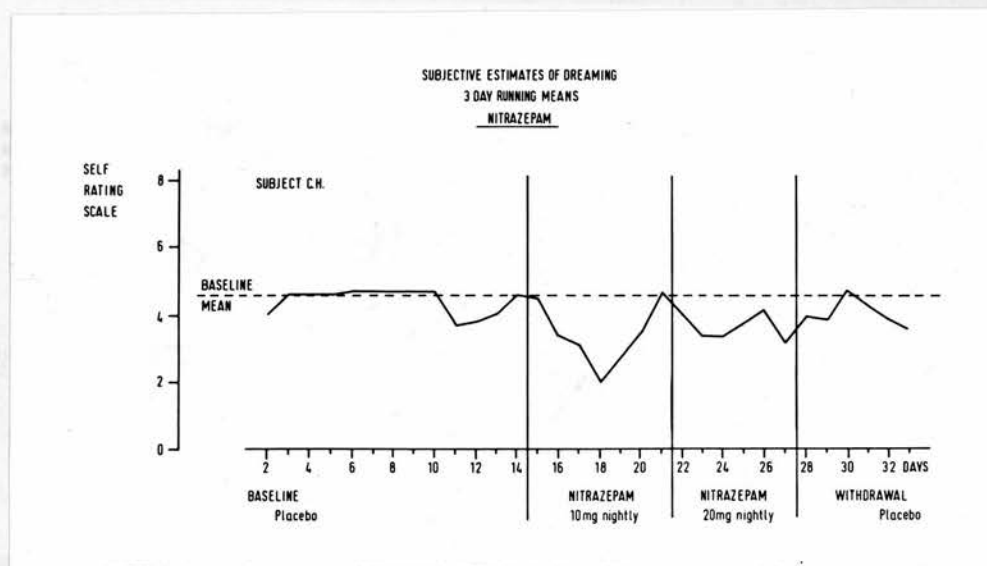


FIGURE 26. EYE MOVEMENT PROPUSION: EFFECT OF NITRAZEPAM.

The effect of the drug is highly significant. Dose effects and withdrawal effects were however not significant. Data from experiment 1.



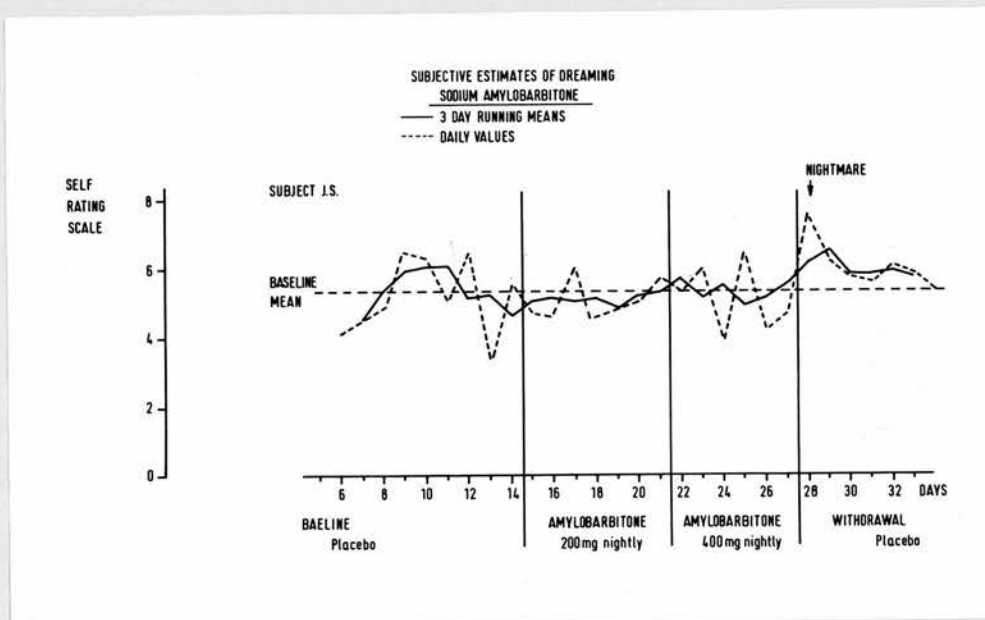
(a)



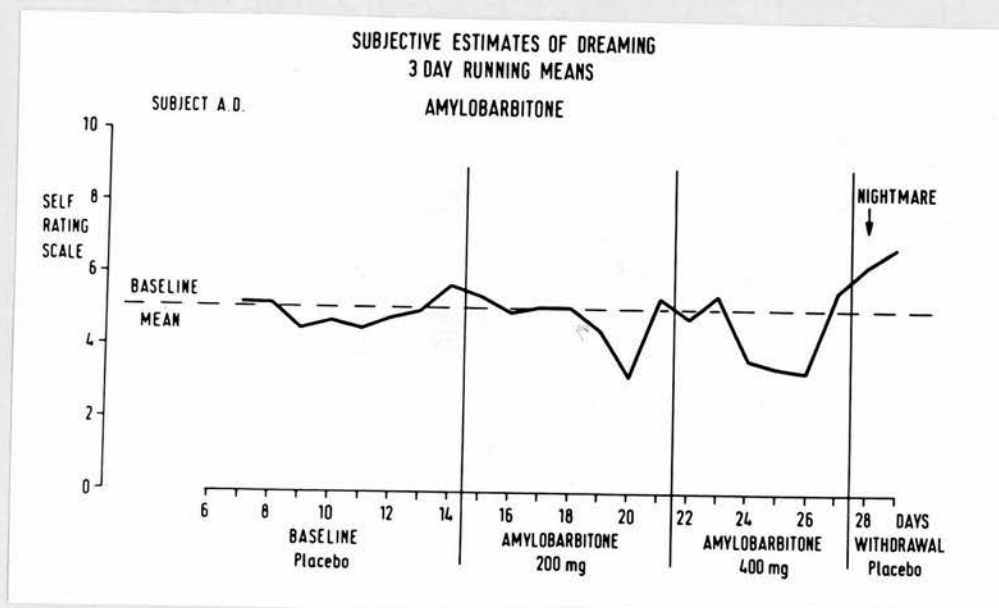
(b)

FIGURE 27. SUBJECTIVE ESTIMATES OF DREAMING: TWO SUBJECTS ON NITRAZEPAM.

- (a) Subject J.T. shows the predicted effects: a subjective depression of dreaming on the drug recovering to normal after some days, and an increase in dreaming during withdrawal.
- (b) Subject C.H. experiences less dreaming while on the drug, but he experiences no increased dreaming during withdrawal. The rating scale used is reproduced in Table 21.



(a)



(b)

FIGURE 29. SUBJECTIVE ESTIMATES OF DREAMING: THE TWO SUBJECTS WHO REPORTED NIGHTMARES.

- (a) Subject J.S. experienced no change in dreaming while on the drug, but had a nightmare when it was withdrawn. Experiment 1.
- (b) Subject A.D. experienced all the predicted changes in dreaming. Experiment 2.

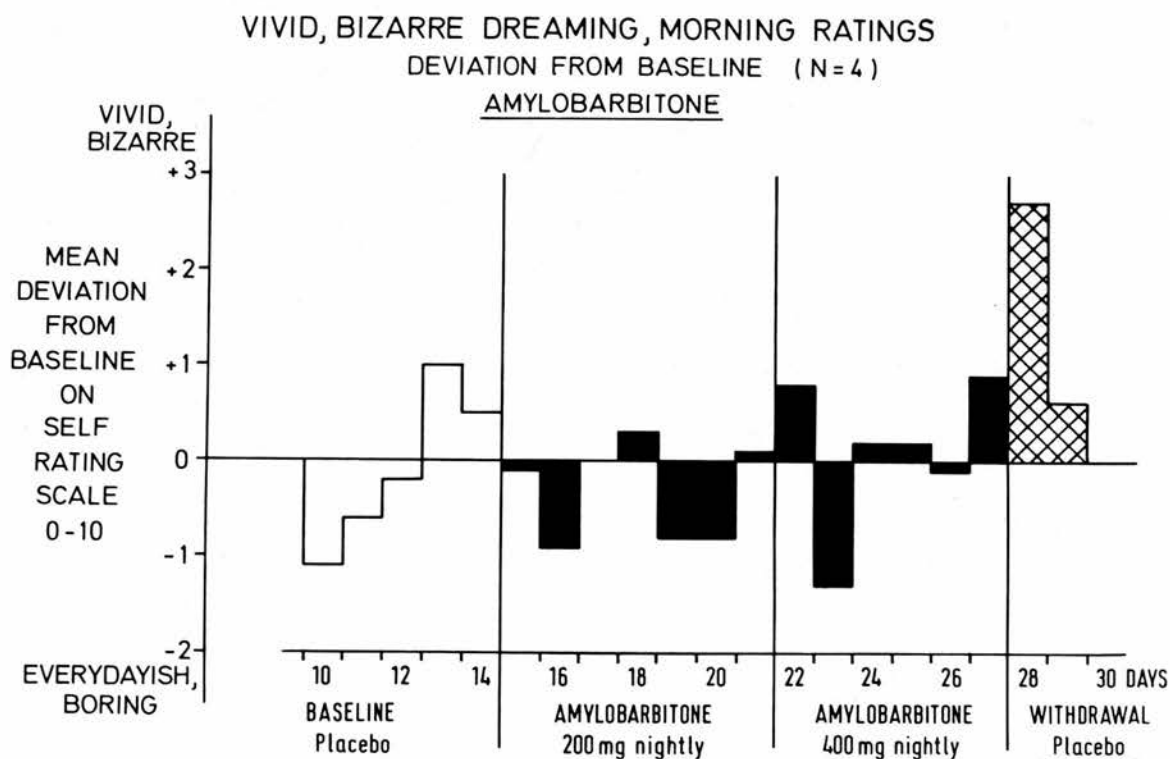


FIGURE 30. SUBJECTIVE ESTIMATES OF DREAMING: "VIVID, BIZARRE" DREAMING AS OPPOSED TO "EVERYDAYISH, BORING" DREAMING.

Means for all four subjects on amylobarbitone in Experiment 2 are presented. Changes during baseline and drug administration are apparently random, but there is a striking incidence of vivid or bizarre dreaming on the first withdrawal night (at home).

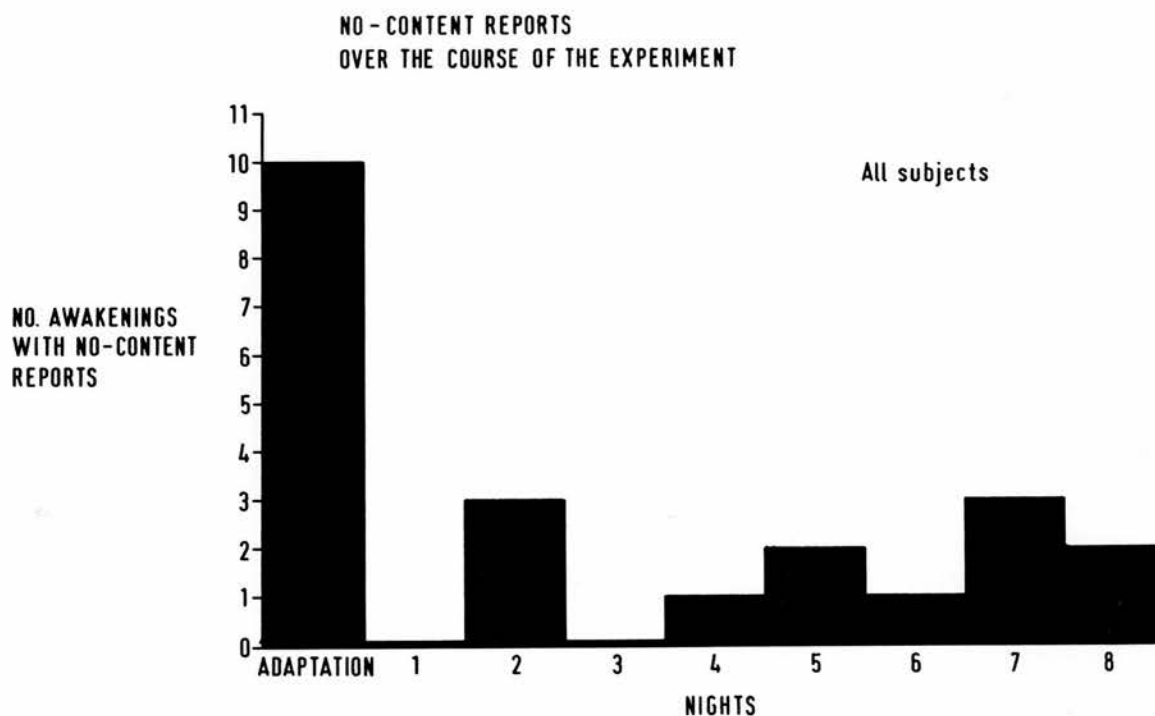


FIGURE 31. THE INCIDENCE OF 'NO CONTENT' REPORTS OVER THE COURSE OF THE FIRST EXPERIMENT.

The total number of awakenings on each night was 24. On the adaptation - to - waking night therefore nearly half of wakenings were fruitless in yielding reports with some item of specific content.

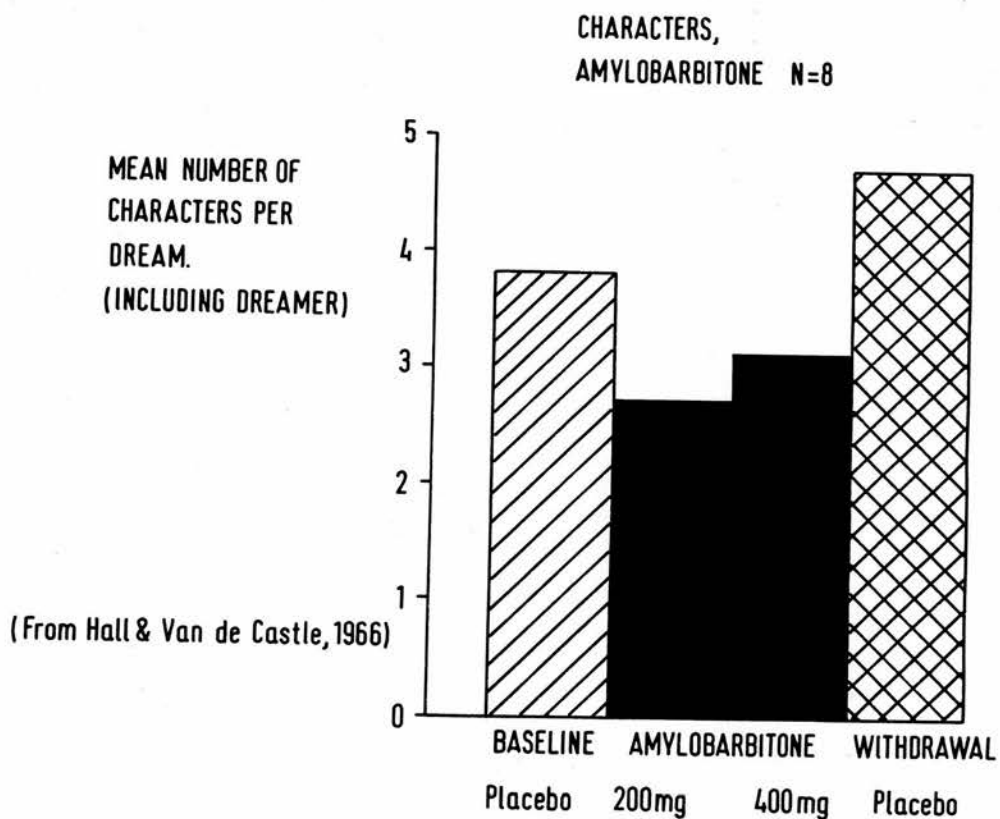


FIGURE 32. THE EFFECT OF AMYLOBARBITONE ON THE NUMBER OF CHARACTERS, AS SCORED BY THE HALL AND VAN DE CASTLE SCALE.

Data from both Experiments 1 and 2.

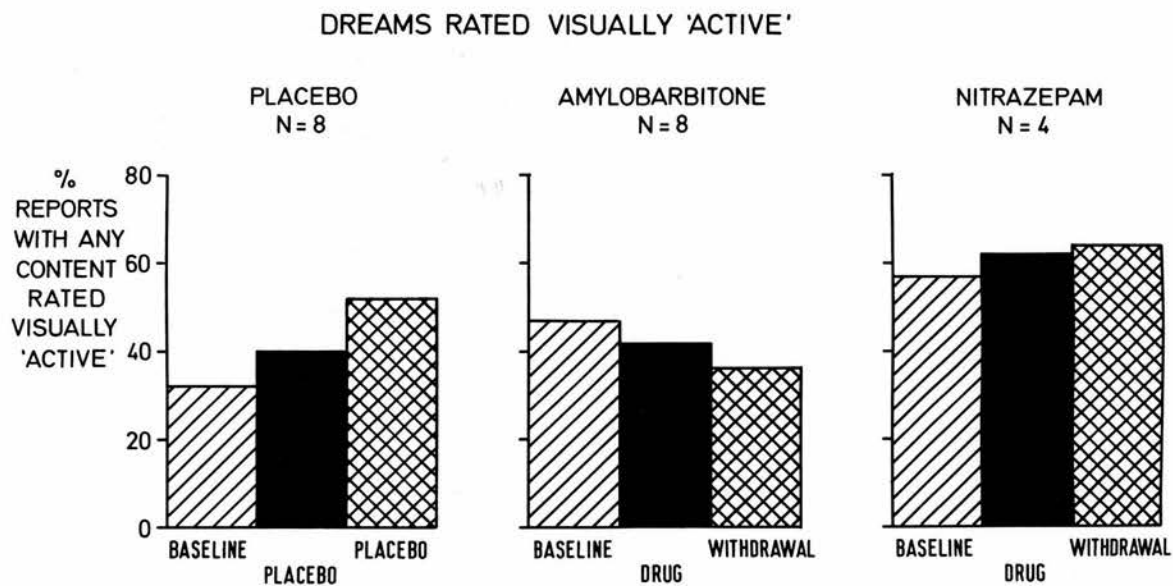


FIGURE 33. RATING OF DREAMS AS 'ACTIVE' OR 'PASSIVE'.

There are no effects of either drug.

EFFECT OF AMYLOBARBITONE ON REM SLEEP
AND ON DREAMS

N = 8

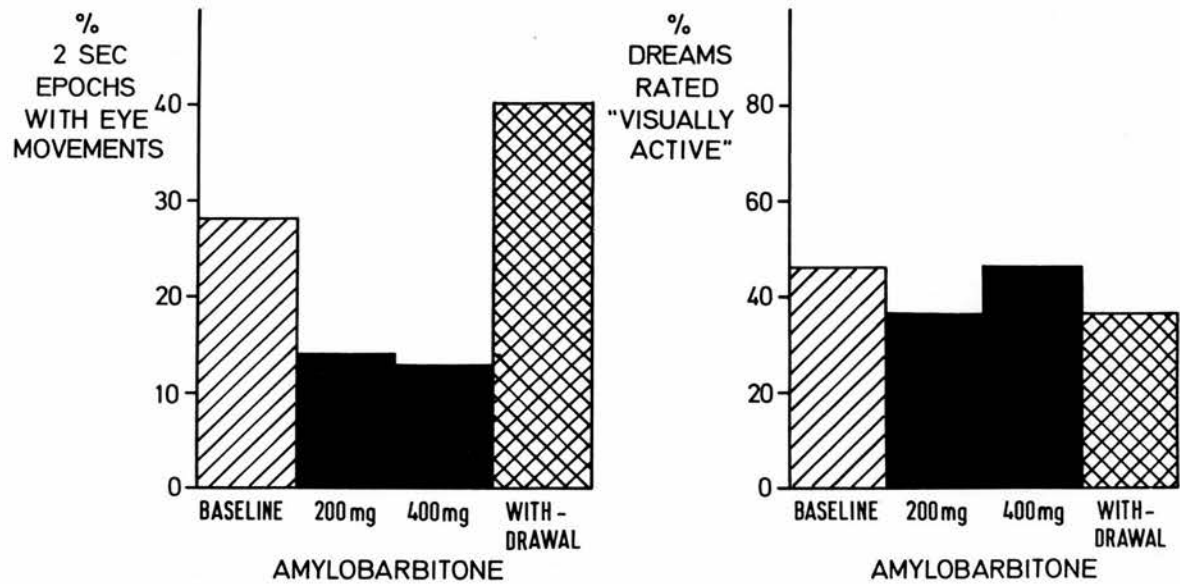


FIGURE 34. CONTRASTING THE EFFECT OF AMYLOBARBITONE ON EYE MOVEMENT PROFUSION WITH ITS LACK OF EFFECT ON DREAM CONTENT.

EFFECT OF NITRAZEPAM ON REM SLEEP
AND ON DREAMS

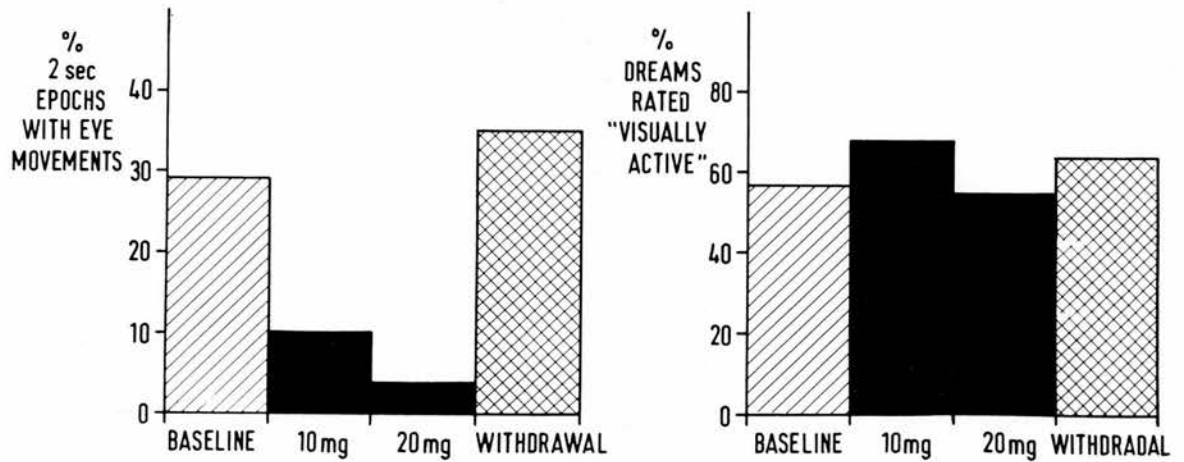


FIGURE 35. CONTRASTING THE EFFECT OF NITRAZEPAM ON EYE MOVEMENT PROFUSION WITH ITS LACK OF EFFECT ON DREAM CONTENT.

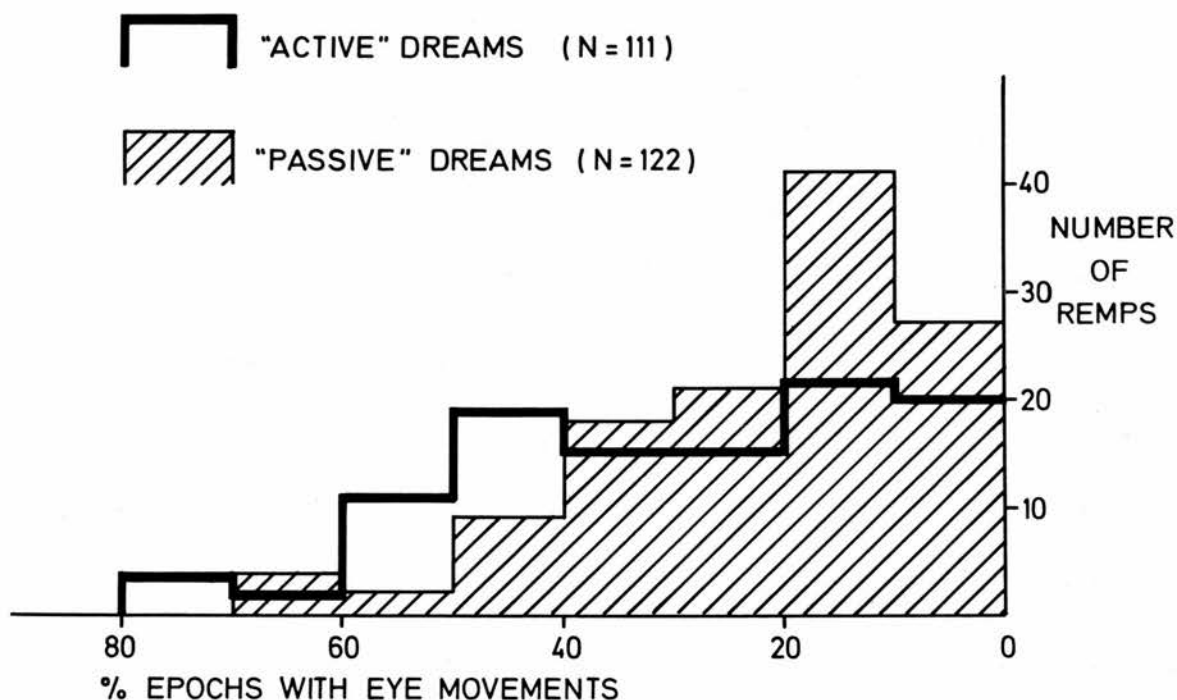


FIGURE 36. PROFUSION OF EYE MOVEMENTS IN THE REMs PRECEDING ACTIVE AND PASSIVE DREAM REPORTS.

Data from all awakenings which produced a report with some content. Data is included from all subjects, whether on placebo or active drug.

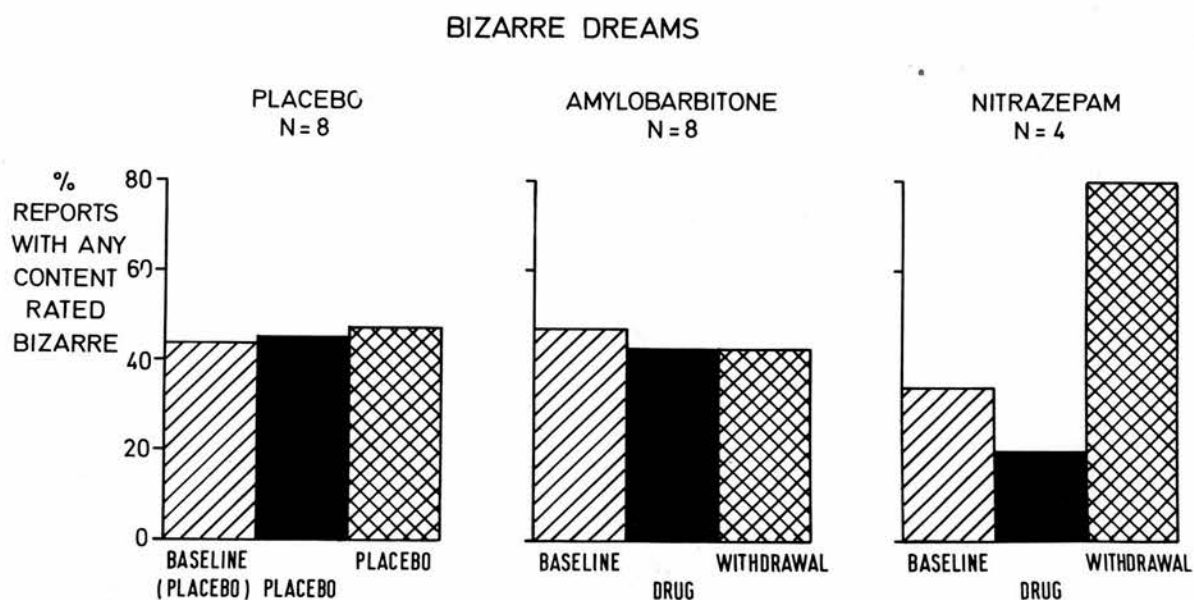


FIGURE 37. THE INCIDENCE OF BIZARRE DREAMS.

Withdrawal of nitrazepam produces a highly significant increase in the frequency of bizarre dreams. Dream reports were rated as bizarre or everydayish according to Foulkes et al (1966).

FOULKES RATINGS AND TIME OF NIGHT

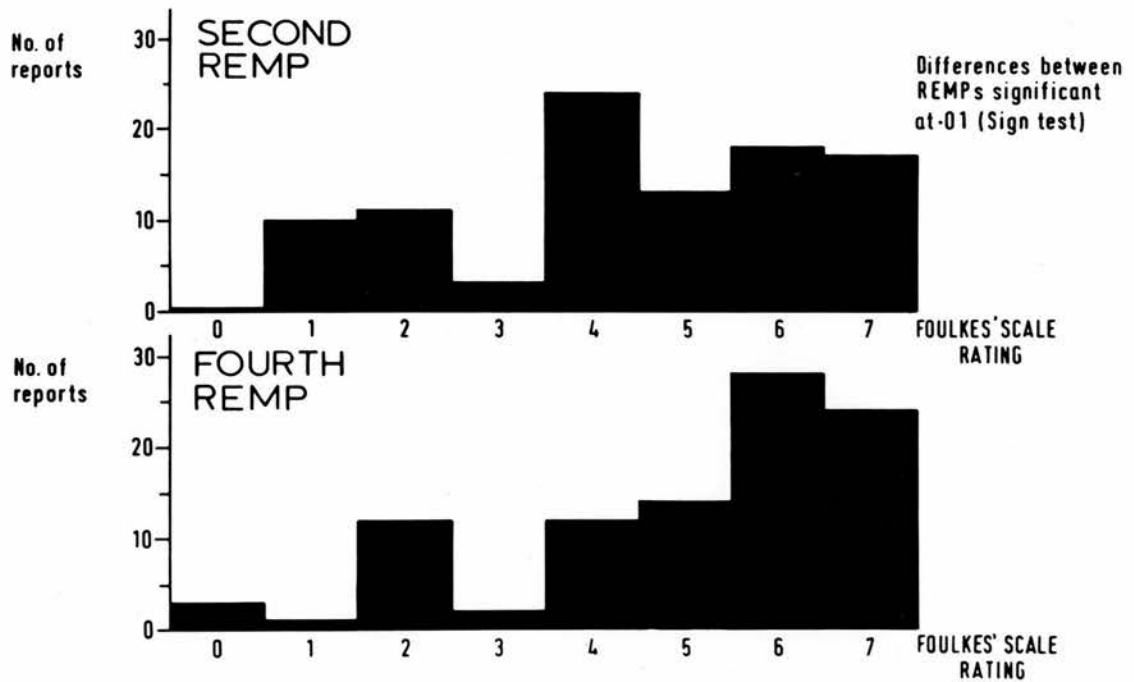


FIGURE 38. THE EFFECT OF TIME OF NIGHT ON DREAM CONTENT.

Data from all experimental nights for all subjects in Experiment 1. The scale (Foulkes et al 1966) extends from 0 - subject believes nothing was happening - to 7 - a visual, bizarre experience which seemed real at the time.

TABLE 25.

LATENCY TO SLEEP ONSET (log transform): Summary of analysis of variance.NITRAZEPAM (Experiment 1):

SOURCE	SS	DF	MS	F	p
Subjects	9.097	3	3.032	14.16	.01
Nights	3.729	7	0.532	2.49	.05
Subjects x Nights	4.501	21	0.214		
Total	17.328	31			

Orthogonal comparisons showed that differences between baseline and withdrawal were significant. Differences between baseline and drug conditions were not significant.

AMYLOBARBITONE (Experiments 1 and 2):

SOURCE	SS	DF	MS	F	p
Subjects	2.857	7	0.408	1.11	ns
Nights	0.535	3	0.178	< 1	ns
Subjects x Nights	7.669	21	0.365		
Total	11.062	31			

PLACEBO (Experiments 1 and 2):

SOURCE	SS	DF	MS	F	p
Subjects	12.903	7	1.843	4.60	.01
Nights	0.425	3	0.141	< 1	ns
Subjects x Nights	8.410	21	0.400		
Total	21.739	31			

TABLE 26.

TIME OF THE EXPERIMENTAL WAKENINGS: Summary of analysis of variance.NITRAZEPAM (Experiment 1):

SOURCE	SS	DF	MS	F	p
Subjects	21.05	3	7.01	5.40	.01
Nights	6.64	7	0.94	< 1	ns
Subjects x Nights	27.26	21	1.29		
REMP	143.70	1	143.70	1217.80	.001
Subjects x REMP	0.35	3	0.11		
REMP x Night	0.42	7	0.06	< 1	ns
Subjects x REMP x Nights	6.72	21	0.32		
Total	206.17	63			

AMYLOBARBITONE (Experiment 1):

Subjects	35.34	3	11.78	27.33	.001
Nights	3.57	7	0.51	1.18	ns
Subjects x Nights	9.05	21	0.43		
REMP	166.41	1	166.41	3961.90	.001
Subjects x REMP	0.12	3	0.04		
REMP x Nights	4.30	7	0.61	4.69	.01
Subjects x REMP x Nights	2.75	21	0.13		
Total	221.57	63			

PLACEBO (Experiment 1):

Subjects	31.28	3	10.42	15.27	.01
Nights	1.07	7	0.15	< 1	ns
Subjects x Nights	14.35	21	0.68		
REMP	167.05	1	167.05	250.40	.001
Subjects x REMP	2.00	3	0.66		
REMP x Nights	0.50	7	0.07	< 1	ns
Subjects x REMP x Nights	6.32	21	0.30		
Total	222.60	63			

TABLE 27.

TIME AWAKE FOLLOWING WAKENINGS: Summary of analysis of variance:AMYLOBARBITONE (Experiments 1 and 2):

SOURCE	SS	DF	MS	F	p
Subjects	658.85	7	94.12	< 1	ns
Nights	1873.54	3	624.51	6.56	.01
Subjects x Nights	1997.32	21	95.11		
REMP	26.26	1	26.26	< 1	ns
Subjects x REMP	716.85	7	102.40		
REMP x Nights	256.54	3	85.51	1.09	ns
Subjects x REMP x Nights	1778.82	21	84.70		
Total	7308.23	63			

PLACEBO (Experiments 1 and 2):

Subjects	3762.18	7	537.45	6.06	.001
Nights	139.06	3	46.35	< 1	ns
Subjects x Nights	1861.68	21	88.65		
REMP	784.00	1	784.00	6.61	.05
Subjects x REMP	830.25	7	118.60		
REMP x Nights	574.87	3	191.62	1.56	ns
Subjects x REMP x Nights	22566.87	21	122.23		
Total	10518.93	63			

TABLE 28.

EYE MOVEMENT PROFUSION SCORES (log transform): Summary of analysis of variance.NITRAZEPAM (Experiment 1):

SOURCE	SS	DF	MS	F	p
Subjects	7.704	3	2.568	4.17	.05
Nights	47.574	7	6.796	11.06	.001
Subjects x Nights	12.898	21	0.614		
REMP	0.097	1	0.097	2.51	ns
Subjects x REMP	0.117	3	0.039		
REMP x Nights	2.020	7	0.288	1.57	ns
Subjects x REMP x Nights	3.858	21	0.183		
Total	74.271	63			

AMYLOBARBITONE (Experiments 1 and 2):

Subjects	12.395	7	1.770	4.32	.01
Nights	19.003	3	6.334	15.48	.001
Subjects x Nights	8.601	21	0.409		
REMP	1.172	1	1.172	2.59	ns
Subjects x REMP	3.170	7	0.452		
REMP x Nights	0.361	3	0.120	1.01	ns
Subjects x REMP x Nights	2.490	21	0.118		
Total	47.194	63			

Orthogonal comparisons showed that for both nitrazepam and amylobarbitone differences between baseline and drug conditions were significant. Differences between doses, and differences between withdrawal and baseline, were not.

PLACEBO (Experiments 1 and 2):

Subjects	10.714	7	1.530	3.08	.05
Nights	0.022	3	0.007	< 1	ns
Subjects x Nights	2.459	21	0.117		
REMP	0.430	1	0.430	2.67	ns
Subjects x REMP	1.132	7	0.161		
REMP x Nights	0.177	3	0.059	< 1	ns
Subjects x REMP x Nights	2.159	21	0.102		
Total	17.096	63			

TABLE 29.

TOTAL CHARACTERS INCLUDING DREAMER: Summary of analysis of variance:ANYLOBARBITONE (Experiments 1 and 2).

SOURCE	SS	DF	MS	F	p
Subjects	102.48	7	14.64	5.28	.0
Nights	34.42	3	11.47	4.14	.0
Subjects x Nights	58.20	21	2.77		
REMP	6.89	1	6.89	< 1	ns
Subjects x REMP	71.98	7	10.28		
REMP x Nights	4.67	3	1.55	< 1	ns
Subjects x REMP x Nights	144.95	21	6.90		
Total	423.60	63			

Orthogonal comparisons show that differences between active drug and withdrawal are significant, but differences between baseline and active drug, and between baseline and withdrawal are not significant.

PLACEBO (Experiments 1 and 2):

Subjects	105.68	7	15.09	1.95	ns
Nights	21.31	3	7.10	< 1	ns
Subjects x Nights	162.43	21	7.73		
REMP	30.25	1	30.25	3.09	ns
Subjects x REMP	68.50	7	9.78		
REMP x Nights	39.12	3	13.04	1.00	ns
Subjects x REMP x Nights	137.12	21	6.52		
Total	564.43	63			

TABLE 30.

POINT BISERIAL CORRELATIONS BETWEEN ACTIVE-PASSIVE RATINGS FOR DREAMS AND EYE MOVEMENT PROFUSION SCORES FOR THE ASSOCIATED REMPS.

Experiment 1.	N (no. dreams)	Mean no. 2 sec epochs with EMs in 5 min.	r	DF	p
Placebo group	59	Active 44.54 Passive 35.34	.190	58	ns
Amylobarbitone group	59	Active 32.04 Passive 23.79	.175	58	ns
Nitrazepam group	57	Active 33.74 Passive 19.18	.264	56	ns
All subjects	118	Active 38.16 Passive 29.65	.174	117	ns
Experiment 2.					
Placebo group	61	Active 60.72 Passive 43.91	.519	60	.01
Amylobarbitone group (all nights)	58	Active 33.95 Passive 31.94	.038	57	ns
Placebo nights	29	Active 47.50 Passive 45.06	.040	28	ns
Drug nights	29	Active 20.41 Passive 18.82	.044	28	ns
All awakenings (excepting adaptation nights and no content reports)	233	Active 40.63 Passive 32.10	.162	232	.05
REMP 2 alone	116	Active 40.11 Passive 28.76	.203	115	.05
REMP 4 alone	117	Active 41.08 Passive 35.38	.114	114	ns

r is significant at .273 with 50 DF, .195 with 400 DF, and .138 with 200 DF.

/withdrawal nights as on any other night (Fig. 20). Neither drug nor withdrawal effects were present in the group who took amylobarbitone, either in experiment 1, or in the combined results for both experiments.

The latency between sleep onset and the first REM of the night was increased by both the drugs. In a non-parametric analysis of variance, this effect was significant for nitrazepam (chi-square = 17.0; with 7 DF, $p < .05$), but did not reach significance for amylobarbitone. However, the mean REM latency was over 100 minutes on all the barbiturate nights, and under 100 minutes on all the placebo nights (Fig. 21). Withdrawal effects (abnormally short REM latencies) were not evident either following nitrazepam, or following the barbiturate (see Fig. 21 again).

The length of REMs from which subjects were woken was analysed to ensure there were no systematic differences which might bias the results; subjects should have got $7\frac{1}{2}$ minutes on every occasion. There were no significant effects. Thus it can safely be said that any differences in dream content between different experimental conditions could not have been due to differences in the length of the respective REMs.

(b) Time of wakenings; Wakingup; Getting back to sleep:

Analyses of the time at which subjects were woken in the night showed, not unnaturally, significant differences between the times of the first and second wakenings (REMPs 2 and 4). In experiment 1 there was in addition a significant REM \times NIGHT interaction in the group/

/group of subjects receiving amylobarbitone (see Table 26). Over both experiments the trend was still present, but was not significant. Examination of the data revealed the origin of this interaction. First, on active drug REMPs occurred later. Second, on withdrawal, REMP 2 occurred early. But subjects then took so long to get back to sleep without their sleeping pill that REMP 4 actually occurred later than it did in the baseline condition. These changes are illustrated schematically in Fig. 22. The tendency of subjects to remain awake a long time on withdrawal from barbiturate is shown in Fig. 23. This effect is significant (see Table 27).

So there were some significant changes in the actual time of night when subjects were woken for their dreams. Since time of night is related to dream content, it is legitimate to ask what effect these changes would have on dream content. On active drug nights the later wakings (on average 40 mins later) would tend to counteract ^{any} ~~the~~ effect of the drug in reducing dream 'intensity'. On withdrawal, however, the opposite effect would occur. Since wakings from the latter half of the night were even later on withdrawal than during baseline (by 1 hour on average), we would expect that combined with any tendency for spicier dreams on withdrawal, the late wakings then would lead to exceedingly active dreams.

There were no significant differences among experimental nights in any of the drug groups in either latency to EEG arousal following the stimulus, or in latency from the stimulus to the subject's first word in answer to "Could you tell me what was passing through your mind?". The only significant effect on either of these variables was a time/

/time of night effect: it took some subjects longer to respond verbally when woken from the second REMP than when woken from the fourth REMP of the night.

(c) The profusion of eye movements:

The effect of the two drugs on the profusion of eye movements was dramatic. The effects are illustrated in Figures 24, 25 and 26, and in Table 28. For both amylobarbitone and nitrazepam the effect of the drug as compared with baseline was highly significant, and cut eye movement profusion to at least a third of baseline levels. A dose effect seems apparent in the figures, with especially low eye movement profusions on the first night with the full dose. However, dose effects and tolerance effects (first to sixth night) were not significant.

A withdrawal effect is possibly suggested by both Figures 25 and 26, but was significant for neither amylobarbitone nor nitrazepam.

3. Subjective estimates:

(a) Anxiety and concentration: Evening ratings for anxiety and concentration showed no changes during the whole experiment: there were neither any detectable effects of the sedatives, nor any detectable effects of their withdrawal.

(b) Subjective measures of dreaming: The striking thing about the subjective measures of dreaming is the great variation in individual response. It had been predicted that subjects would report decreased dreaming on the active drugs, with a 'rebound' increase on withdrawal. Some subjects in fact reported no change; others reported decreased dreaming on the drug, but no 'rebound' subsequently; yet others/

/others reported an unusual amount of dreaming in the withdrawal phase (two subjects reported nightmares¹ at home), but experienced no changes while actually on the drug. One subject reported some very vivid, aggressive dreaming on amylobarbitone. A selection of different subjective responses to the two active drugs is illustrated in Figures 27, 28 and 29. Note that only two subjects (Figs 27a and 29b) actually experienced both an effect of the active drug and a withdrawal effect. The two nightmares¹ were both reported at home on the first withdrawal night. They are given in Appendix IV; also in the appendix is quoted one of the very vivid, aggressive dreams reported by one subject while on 400mg of amylobarbitone.

In the second experiment, subjects were in addition asked to try to differentiate between the length or time they felt they had spent dreaming, and the "intensity" of the dreaming. Whether because subjects found it difficult to discriminate, or because the two measures in fact varied together, this discrimination did not seem possible in practice. Estimates of 'time spent dreaming' and of 'vividness' of dreaming both varied in like manner. Estimates of 'time spent dreaming' correlated highly with the global estimates of dreaming: Spearman's correlation coefficient was 0.93 when the mean for the four amylobarbitone subjects was compared for each of 22 days. Subjective estimates of vividness of dreaming did not correlate quite so highly with the other two measures: correlations of 'vividness' of dreaming with the 'time spent dreaming' and with the global estimate were both 0.82.

When the distinction was attempted, this was done in the hope/

¹The term nightmare is used here exclusively to refer to the subject's experience. No objective criterion is used. The term is only employed when the subject himself called his experience a nightmare. Such experiences are usually but not necessarily rated very high for vividness etc., on the self-rating scales.

/hope that while subjective experience of time spent dreaming would probably reflect the proportion of the night spent in REM sleep, 'vividness' would provide a measure of subjective intensity of dreaming on the nights when subjects were sleeping at home. It may be that distinguishing these two elements from a night of sleep at home is not possible. However, in so much as the measures do not correlate perfectly, and the measure of subjective 'intensity' of dreaming is valid, it would appear that 'intensity' of dreaming, as judged by subjects the following morning, was altered during the experiment. This is illustrated in Figure 30. The figure shows a marked effect on the first night of withdrawal, for the four subjects in experiment 2 who received amylobarbitone. One of them reported a nightmare¹ then, but all reported a marked increase in vividness of dreams that night. Interestingly, there is a withdrawal effect, but apparently no effect of administration of the active drug itself.

(4) The dreams

(a) No-content reports: The number of no-content reports obtained in experiment 1 is illustrated in Figure 31, for each night of the experiment, for all subjects. A no-content report is defined as one with no item of specific content. The number of no-content reports thus includes cases where the subject thought nothing had been happening (which were very rare), as well as cases where the subject thought something had been happening, but he could not recall it.

The number of no content reports was unrelated to either drug, or to the REMF from which the subject was woken. There was/

¹see footnote on page 229.

/was however the dramatic effect illustrated on the adaptation night. This figure demonstrates that one night of adaptation-to-waking was indeed necessary, and also that one such night (in addition to the adaptation-to-laboratory) was sufficient. Because of this effect all data from the adaptation-to-waking night was ignored in the further analysis of the data.

Because no-content reports were uniformly distributed throughout experimental nights in all groups of subjects, no attempt was made to exclude them from the calculations involving analysis of variance. They were treated as reports with no characters, and no activities, and a verbal length of zero. No-content reports were in any case marginally too frequent to allow interpolation to be reliable. For the statistical analyses not involving analysis of variance however (the Foulkes' scale, active/passive ratings etc.) the no-content reports could simply be excluded from the calculations.

(b) Length of the verbal report: The length (number of words) of the reports was unaffected by either drug, or by time-of-night, whether raw word-length, or edited word-length is considered. Indeed analyses of variance revealed no significant effects besides between-subject differences. For this reason no correction for report length was applied to any of the Hall and Van de Castle scales.

(c) Results of the Hall and Van de Castle system of content analysis:

The analyses of variance revealed surprisingly few effects apart from significant differences between subjects, which were very prevalent. In the first experiment, the only significant effect apart from/

/from between subject differences occurred in the number of unfamiliar characters in the placebo group: there was a significant difference between nights. However, since there was no trend to these differences across nights, and since this was the only statistically significant effect in over a dozen analyses, no weight is placed on this finding. This effect was not present in the combined data of experiments 1 and 2.

However, when the results for the larger number of subjects were analysed (combining the data for experiments 1 and 2), there were two meaningful effects. The first was that the total number of characters including the dreamer differed significantly among nights in the group taking amylobarbitone, but not in those only getting a placebo. The number of characters per report was decreased relative to baseline by both doses of the drug, and was above baseline on withdrawal. Only the active drug - withdrawal differences were significant, however. These changes are illustrated in Figure 32 and in Table 29.

The second finding from the larger experiment was an effect of REMP on the number of unfamiliar characters. This effect was significant in the eight subjects taking placebo, and although not significant in the other eight subjects on amylobarbitone, the trend was in the same direction: for more unfamiliar characters in the dreams collected from REMP 4 than in those from REMP2.

There were no significant effects apart from between-subject differences in the total number of activities, in either experiment. Nor were there any effects of either drug upon the total number of/

/of activities performed by the dreamer. There was however an effect of nitrazepam on the number of dreams involving some looking or watching activity on the part of the dreamer. Nitrazepam significantly decreased the number of such dreams, and there was an increase in their frequency on withdrawal ($\chi^2 = 9.37$, $DF = 3$, $p < .05$). Amylobarbitone had no such effect. However, it is important to bear in mind that the number of dreams with visual activities on the part of the dreamer is not large: only one third of all dreams included any looking or watching activities by the dreamer. Further, an examination of the mean eye movement profusion scores for the REMPs yielding dreams with and without visual activity by the dreamer showed no differences in profusion of eye movements.

The number of social interactions was so small (half the subjects averaged less than one interaction per dream) that excepting comparisons between REMPs, analyses were made on the number of dreams with social interaction. Since only two sexual interactions were encountered in nearly 300 reports, these were combined with friendly interactions. This lack of sexual interactions is striking in comparison to Hall and Van de Castle's norms (see Table 32). They report 70 sexual interactions occurring in 58 out of 500 dreams collected from men. The lack of sexual interactions in this experiment is almost certainly a function of the fact that these dreams were collected under laboratory conditions (Hall and Van de Castle's were home dreams) - the laboratory probably inhibits the actual occurrence of sexual dreams as well as possibly inhibiting subjects from reporting them when they occur.

Other norms from this study are given in Table 22, and compared with Hall and Van de Castle's.

There were no effects of either drug on the number of dreams with either aggressive or friendly interactions, or on the number of dreams in which the dreamer was an active participant in social interaction.

The only variable to have a discernible effect on social interactions was time of night: a sign test revealed a significant tendency for more interactions in dreams from the fourth REMP than from the second REMP ($p < .01$).

The number of dreams with emotion, success or failure, misfortune or good fortune was quite unaffected by either drug treatment, or even by the time of night. Physical surroundings and descriptive elements were also unaffected by either drug. There was a significant effect of time of night upon the number of negatives used in the dream report: more were used later in the night. Unedited report length was (not significantly) greater for awakenings later in the night, which may shed light on this otherwise curious finding.

An analysis of the dramatic intensity index, using a Friedman two-way analysis of variance by ranks on the means for each night (i.e. averaging results from the two wakings) failed to show any effect of either drug: this index proved no more useful than the original scales of Hall and Van de Castle it was derived from.

The "Quality" hypothesis:

Predictions made on the basis of the 'quality' hypothesis, that the sedatives would make dreams pleasanter, proved essentially negative. As already described, there were no effects of the drugs on the number of emotions, negative or positive, no effects upon the number of aggressive, or friendly interactions, no effects upon the numbers of familiar or unfamiliar characters, of good fortunes or misfortunes, of successes or failures, or on the numbers of negatives used in the reports. The number of positive and of negative instances of the evaluative subscale of descriptive elements showed no evidence of the effect of any variable. Lastly, combining friendly and sexual social interactions, happiness, success and good fortune into one 'positive quality index', and aggressive interactions, negative emotions, failure and misfortune into a 'negative quality index' proved equally fruitless.

However, in so much as the two drugs had differing effects on the dreams (nitrazepam making them everydayish, while amylobarbitone did not), the few effects of the drugs are more consistent with this hypothesis than with any other. Clearly these effects cannot be understood as effects on the 'intensity' of dreaming, nor can they be understood as being mediated by effects on EM profusion.

(d) The psychodynamic scales of Whitman et al: There were no effects of either drug on Hostility, Anxiety, or Heterosexuality as measured by these scales; nor were there any effects of time of night on these measures. This was true both for the raw scores and for the scores corrected for length of the verbal report.

(e) The whole-dream rating scales:

(i) Active/passive ratings: At the end of the first experiment there appeared to be an effect of amylobarbitone on the proportion of active as

/There was clearly no effect of nitrazepam.

When the results for all eight subjects from both experiment 1 and 2 were available, however, it became clear that the effect of amylobarbitone was illusory: there was in fact no effect of either drug (Figure 33) ($\chi^2 < 1$).

Figures 34 and 35 illustrate dramatically on the one hand the marked effect of the two drugs on the profusion of eye movements, and on the other the total lack of any effect on the proportion of active dreams. While the two sedatives drastically reduce the phasic 'activity' of the REMPs, there was no concomitant effect on the 'activity' of the dream reports.

The original prediction that these two drugs would alter the proportion of active versus passive dreams was made on the basis of the relationship described by Dement and Wolpert (1958) and by Berger and Oswald (1962c), in which they found a significant association between REMPs judged 'active' in terms of eye movement profusion, and the corresponding dreams. From Figures 34 and 35, it was expected that a similar direct test on the data from this experiment would prove negative. The results were not however immediately conclusive. Dividing the REMPs into two groups, those with high and low numbers of 2 sec epochs with eye movements, and using contingency tables, as did Berger and Oswald, revealed no relationship at all. Point biserial correlations between the actual number of epochs with eye movements in the REMP and active and passive dream report categories revealed non-significant associations for each of the three groups of subjects/

/subjects in experiment 1 (placebo, and two drug groups).

The correlation for the nitrazepam group was however very nearly significant. Data from all 12 subjects in experiment 1 was hence pooled: the correlation was again nearly but not quite significant, with active dreams coming from REMPs with slightly higher EM profusion scores.

When experiment 2 was complete, the correlation was tested on all eight subjects receiving placebo in both experiments, and a significant correlation was established. However, the correlation for all the subjects who got amylobarbitone was nearly zero. This appeared to suggest that administration of the drug destroyed an association present otherwise. For the subjects in the amylobarbitone group therefore, separate analyses were made of the placebo nights (baseline plus withdrawal) and the active drug nights, but the correlation between active dreams and high EM profusion was equally low for both conditions.

In the end, data from every waking producing a report with some content from all except the adaptation nights for all 20 subjects were pooled. This expressed the association between EM profusion and the dream content for all experimental nights throughout the experiments. Active dreams were associated with higher EM profusion scores, and this correlation was significant. The correlation coefficients, and mean EM profusion scores are given in Table 30. When the distributions of EM profusion scores are plotted for the REMPs resulting in active and passive dreams respectively (Figure 36) it is clear that there is a difference between these two distributions: a lot of passive dreams/

/dreams come from REMPs where only 10-20% of epochs contain eye movements.

On the other hand it is quite clear from Figure 36 that there are many active dreams whose REMPs contained almost no eye movements at all.

It has been argued (Hauri and Van de Castle, 1972) that the association between active and passive dreams and EM profusion may be solely dependent upon the fact that EM profusion is higher later in the night, and also mental reports are more 'active' from awakenings later in the night. There was indeed a significant time of night effect upon the proportion of active dreams in the first experiment - there being more active dreams from REMP 4 awakenings than from REMP 2 awakenings. (Sign test, $p < .05$). The same trend was present in the second experiment, but was not significant. Although not significant, there was also a consistent trend for slightly higher EM profusions in the fourth than in the second REMP. Therefore, the association between active/passive ratings and EM profusions was tested separately for each REMP. Within awakenings from REMP 2 the correlation between active/passive dreams and EM profusion is still significant. Although not significant within REMP 4 awakenings it is still positive. Thus it seems reasonable to conclude that the association does hold up independently of the time of night effect. However, it should also be quite clear from Figure 36, and from this discussion that the association is a tenuous one: it is not the dramatic effect one might have believed from earlier reports.

- (ii) Ratings for sexiness, anxiety and psychotic thinking: These ratings showed neither any effect of the drugs, nor any effect of time of night. The finding reported in Carroll et al (1969), that dreams with sexual themes predominated in withdrawal, was not confirmed.
- (iii) Blind judgements: Blind judgements by the experienced judge as to whether dreams came from baseline, drug or withdrawal conditions were unsuccessful. At the end of the first experiment the judge was in fact (insignificantly) worse than chance! Taking both experiments together his judgements were no different from chance (chi-square less than 1).
- (iv) Foulkes dreamlike fantasy scale: Because this scale effectively combines several "factors", analyses of the results were made separately for each "factor". Reports with some specific content are distinguished from those with none; as has been mentioned earlier, no content reports were distributed evenly throughout all nights (excepting the adaptation night). Reports with perceptual (i.e. visual) content are distinguished from those which are only conceptual. Dreams which were 'real' at the time (hallucinatory) are distinguished from those containing an element of awareness that the events are only a dream. Lastly, a separation is made between everydayish and bizarre reports.

The only one of these "factors" to show any effect of the experimental manipulations was that of bizarreness versus everydayishness. In the first experiment the placebo group showed an increase in the proportion of bizarre dreams across the eight experimental nights which was significant at the .05 level; however, when the results for all eight placebo subjects from both experiments were analysed, this/

/this effect was no longer evident (see Figure 37). There were no changes in the proportion of bizarre dreams in the amylobarbitone group in either experiment.

The effect of nitrazepam was however quite striking: the proportion of bizarre dreams drops on the drug, but increases dramatically on withdrawal. The proportion of bizarre dreams (of those with any content) was 33% on baseline, 31% on 10mg, 7% on 20mg nitrazepam, and 79% on withdrawal. Differences between baseline and drug conditions are not significant, nor are differences between the doses, but differences between withdrawal and baseline, and withdrawal and drug nights are significant (chi-square = 4.31 and 11.50, $p < .05$ and $p < .001$ respectively).

(f) Time of night effects: Time of night effects (differences between data from different REMPs) are scattered through the results. Frequently differences occurred consistently in all groups of subjects and in both of the experiments, but did not reach significance in all groups. For instance, there was a higher proportion of 'active' dreams from awakenings later in the night in both experiments 1 and 2, but this result was only significant in the first experiment ($p < .05$, sign test). One would not wish to claim with certainty that such an effect exists therefore, but I am inclined to believe it to be a genuine effect.

Besides this result, there were a variety of measures where differences in either experiment 1 or 2 occurred as a function of time of night in the subjects receiving placebo - i.e. where we can discount any interaction with drug effects. Such measures included the/

/the number of negatives used in the verbal report ($F(1,3) = 10.67$; $p < .05$), the number of unfamiliar characters ($F(1,7) = 7.72$; $p < .05$), and the number of social interactions ($p < .01$, sign test). The time spent awake after awakenings was also longer later in the night ($f(1,7) = 6.61$, $p < .05$).

Curiously one of the variables which one would have expected to be most sensitive to the effects of time of night, namely the profusion of eye movements, did not show a significant effect, although there were more epochs with eye movements in REM 4. With the analysis of variance used, very high F ratios were needed to make REM effects significant.

Figure 38 illustrates how the Foulkes' dreamlike fantasy scale scores differed significantly between early and late awakenings in the first experiment ($p < .01$, Sign test), though once again in experiment 2 the same trend did not reach significance. It is noticeable that more early night reports are scored 4 or 5 on the scale, indicating some degree of awareness by the subject that he was dreaming. A simple test of the effect of time of night on REM reports using Foulkes' scale has not previously been reported though Pivik and Foulkes (1968) did find a similar time of night effect with NREM reports.

CHAPTER EIGHTEEN

DISCUSSION

The results of this study were not as expected. They did not confirm the results of an earlier study (albeit a small study) in this laboratory by Carroll et al (1969). It had been assumed that there was some relation between eye movements and dreaming. Both the scanning hypothesis and the intensity hypothesis had predicted that, given the drastic effect of the drugs on the profusion of eye movements, dream content would have been markedly affected by the drugs. The data has failed to confirm either of these hypotheses.

The results have not proved that there is no association between eye movements and dream content. Indeed, a slight (and significant) relation was found between eye movement profusion and active dreaming. Moreover, it is logically not possible to prove that an independent variable has no effect on a dependent variable. The essence of the results, however, was that the magnitude of any effects of the drugs on dream content was nowhere near what had been predicted.

The results do not argue that there is no relation between rapid eye movements in REM sleep and dreaming; but they do argue that there is very little relation between the two. The results do not imply that dream content is quite independent of the physiology of the REM, but they do seem to imply that dream content is largely independent of the profusion of EMs in REMs. I shall discuss the results with this last statement in mind.

1. Physiological effects of the drugs:

To start with the physiological effects. The drugs didn't act as sleep inducers, though getting to sleep was difficult after nitrazepam was withdrawn. Since the drugs were taken immediately prior to lights-out, this lack of effect needs little comment - our subjects were not insomniacs to start with. That only nitrazepam significantly delayed the onset of REM sleep is more surprising, since Haider and Oswald (1971) found an equal effect of both drugs. However, though not significant, mean REM latency was 100 minutes or more on both drugs and less than 100 in all placebo conditions. Further, the mean REM latency on withdrawal from the barbiturate was markedly low (Fig. 21), and the only occasion with a latency less than 40 minutes occurred under this condition. One latency of 308 minutes/

/minutes from a subject who spent much of that time drifting between drowsiness and stage 2 sleep on barbiturate withdrawal was no doubt responsible for the barbiturate effect not being significant.

The effects of the drugs on eye movement profusion was marked and as expected. Any lack of effect of the drugs on dream content cannot be ascribed therefore to a lack of change in the "independent" variable. The only indications of dose effects were present here. There appeared to be a slight development of tolerance to the heavier dose of amylobarbitone (Fig. 25), and consistent with this tolerance effect, a slight withdrawal effect was also evident following both the drugs. Though not significant, it is noteworthy that EM profusion was above 30% of epochs only after drug withdrawal (Figs. 25 and 26).

2. The dreams:

There were very few effects of the drugs on dreams. Drug effects were not totally negative, however. Besides the effect that nitrazepam had on the number of bizarre dreams, and on the number of dreams with visual activity on the part of the dreamer, amylobarbitone reduced the number of characters in the dream reports. Since over 20 analyses of variance were performed, it would be expected on a purely chance basis that at least one would yield significant effects. While it is difficult to estimate accurately the number of significant effects to be expected, since the analyses for characters, unfamiliar characters, male characters, etc. are clearly not independent; nevertheless it might seem reasonable to ascribe this effect to chance. It would

/would certainly improve the consistency of the results if we could ascribe the reduction in characters on amylobarbitone to chance. However, the effect was in the predicted direction, and more important, this reduction was present at both doses, with an increase on withdrawal (see Figure 32). Because I am interpreting the bulk of the results as showing no effect of the drugs, I think it would be improper to reject this result as being merely a chance one. Whatever the interpretation of this result, at least I think it is clear that this isolated drug effect on its own cannot be interpreted as supporting a "scanning" hypothesis for rapid eye movements: nor, in the absence of any effects of the drugs upon the number of activities or social interactions, can it be taken as support for an "intensity" hypothesis.

The results from the Foulkes' dreamlike fantasy scale are important in this connection. Breaking this scale into its component dichotomies of visual/conceptual, real-at-the-time (hallucinatory), or not, everydayish or bizarre, the only effect of either drug was on the number of bizarre dreams. Nitrazepam, a tranquillizer, as well as a hypnotic, reduced the number of bizarre dreams; it made them more everydayish. It did not affect the number of perceptual dreams (with visual imagery). This effect is one which I interpret as an effect of the drug on thought-processes, not one which is mediated by the number of eye movements the dreamer makes in scanning his environment. But eye movement bursts in REM sleep are associated with PGO spikes. Using PIPs as an index of PGO spiking in the human (this index is probably closer/

/closer to PGO activity than are EM bursts), Rechtschaffen and co-workers have shown that PIPs are related to the appearance of the novel, the bizarre, in the dream (Watson, 1972 ; Rechtschaffen et al 1972) whether the mentation is in REM or NREM sleep. Maybe then the effect of nitrazepam on the number of bizarre dreams is not via its effect on the EMS of REM sleep so much as via its effect in dampening the more fundamental phasic activity of sleep, PGO spikes.

The effect of nitrazepam on the number of dreams in which the dreamer engages in visual activity does appear to support the scanning hypothesis. Foulkes et al (1972) found that in children's dreams, visual activity on the part of the dreamer was significantly more frequent in dreams collected from 'phasic' awakenings following bursts of eye movements, and they interpreted this finding as supporting the scanning hypothesis. However, in this experiment amylobarbitone had no effect on the dreamer's visual activity, despite a marked effect on the profusion of eye movements. This suggests that the reduction in visual activity by the dreamer under nitrazepam was not a direct result of the reduction in eye movement profusion. Moreover, eye movement profusion was in fact no different in REMs producing dreams in which the dreamer showed visual activity, and in those in which he did not. I believe it is more reasonable to suppose that this effect of nitrazepam is a 'side-effect' of the generally tranquillising effect of nitrazepam on mental activity. This interpretation makes sense if dreaming is thought of as mental activity, instead of being regarded as secondary to the rapid eye movements of REM sleep.

3. Active and passive dreams, and eye movements:

One of the major predictions of the experiment, that drug dreams would be visually "passive" rather than "active", was refuted. This prediction was made on the twin assumptions that the drugs depressed the profusion of eye movements, and that there was a correlation between REMPs high in eye movement profusion and visually "active" dreams. The drugs depressed the eye movements, but not the active dreams.

That active dreaming persists in the absence of the phasic events of REM sleep argues strongly against the idea that it is the phasic events of sleep which are responsible for dreaming. Thus Dement's idea (1969) that PGO spikes are responsible for the occurrence of dreaming both in REM and NREM sleep (which does contain a certain amount of PGO activity) is not supported. The results of these experiments suggest that the presence or absence of dreaming is not related to the occurrence of phasic activity.

Dement (1969) proposed that the primary function of REM sleep is to facilitate the 'discharge' of phasic events. The experiments here cannot offer evidence either in support of or against that hypothesis. Dement however also proposed that if 'discharge' of phasic events in REM sleep were suppressed, there would result an enhancement of drive-oriented or emotional behaviour. If the presence of activity, and emotion, in dream content is any guide to the strength of drive-oriented behaviour, then no increase was observed here, under the conditions when EM activity was much reduced. On the assumption that EM burst activity is some guide to the incidence of PGO activity, then Dement's hypothesis that PGO activity helps 'discharge' drive is not supported. (This last deduction from the results is however rather speculative).

The results here might appear to argue against Molinari and Foulkes' idea (1969) that phasic events are crucial to the sort of content of mentation derived from either REM or NREM sleep. Molinari and Foulkes' finding that "PVE" (primary visual experience) was associated with the presence of eye movement bursts might appear to be inconsistent with the present results. This, however, would arise from a misunderstanding of the nature of their findings. Especially in their replication of the original result, (Foulkes and Pope, 1972) they stress that both PVE and SCE (secondary cognitive elaboration) can be found in most REM reports. The critical point is that the occurrence of EM bursts 'suppresses' SCE: EM bursts do not create visual experience, they merely make the visual experience primary.

After Molinari and Foulkes' original finding had been confirmed in this laboratory by Michael Holmes, an attempt was made to rate some of the dreams in this experiment for SCE. No effect of the drugs was evident in a small sample of dreams, but not much weight should be put upon this result. These awakenings were not designed for discriminating SCE from PVE, and the judges did not find it easy to adapt Molinari and Foulkes' scoring system to apply to whole dream reports instead of to the very last experience.

Given the complete absence of any drug effect on the number of active dreams, why the overall correlation between active dreams and EM profusion in the REMPs? This does at first seem a very paradoxical finding. It certainly is paradoxical if one thinks of the results/

/results in the following framework: EMs and active dreams are related, drugs reduce EM profusion - so why don't they affect dreams? What is more important about the correlation between EM profusion and active dreams is not its significance, but its magnitude. There is a tendency in psychological research to concentrate upon the statistical significance of findings to the exclusion of a sense of proportion in assessing the meaning of the results. All too often in discussion of research findings, the statement that a result was 'significant' is taken as the last word. The magnitudes of correlations are ignored (see Bakan, 1966, for a full discussion of this). The correlation between active dreams and EM profusion in these experiments was .16. This correlation was significant, but it is small. It could be expressed another way by saying that of the variance in EM profusion scores, only 2.5% is accounted for by the active or passive nature of the dream. This is a very weak relation. It makes it difficult to argue that the 'intensity' of a dream has such relation to the 'intensity' of the REM. It suggests a very peripheral connection between the two. What is striking in Fig. 36 is the considerable number of active dreams which come from REMs with very few eye movements at all. An illustration of such an active dream is given in Appendix V.

4. The divergence of these results from those of Carroll et al:

Carroll et al (1969) found that barbiturates significantly reduced the proportion of active dreams, significantly reduced the Foulkes' dreamlike fantasy scale ratings, and on withdrawal gave rise to/

/to 'sexy' dreams. This study confirmed none of these results. Only three dreams considered sexual in these occurred in Carroll's study, and all occurred on withdrawal nights. It would not be too surprising if Carroll's finding that sexual dreams occurred only in withdrawal were a chance effect, considering the small number of these dreams (one can roughly estimate this probability as one chance in 30). The other differences between his findings and these results need more comment, however.

To try to tease out the differences between his study and this one, recourse was made to Carroll's raw data (Carroll, 1969). Carroll had examined the profusion of EMs in his study, and a correlation of EM profusion with active/passive dreaming was calculated from his data. This proved to be .35 (35 D.F., $p < .05$). This is a higher correlation than I obtained, but it also lies just within the bounds of statistical significance. The critical factor of his results was that not one of his nine barbiturate dreams was rated as active. Five of the nine baseline dreams and three of the nine withdrawal dreams were rated active. Only one of the nine post-withdrawal placebo dreams was rated active. These last were collected on average nine days after the first withdrawal night. Data from these nights were not included in the published results. The low frequency of active dreams on this final placebo night makes the results from the drug night seem a little less dramatic; there were in fact no significant differences between the drug night and the final night either in the proportion of active dreams or in the scores on Foulkes' dreamlike/

/dreamlike fantasy scale. This last observation, plus the greater number of awakenings in the present experiment, inclines me to trust my own results the more. There are certainly no other differences between these two studies which seem capable of resolving our differences over the effect of the barbiturate on the 'activity' of the dream. Differences in methodology are small: Carroll gave subjects 10 minutes REM before waking them, woke them from REMs 2, 3 and 4, and used Foulkes' interview schema. It is possible that my use of an unstructured interview could have been critical in my failure to find any effects on the dreamlike fantasy scale, but it could hardly explain our differences over active versus passive dreaming.

Carroll et al found a significant reduction in dreamlike fantasy scale ratings on barbiturate. They interpreted this finding to mean that barbiturates made dreams "more conceptual than perceptual, more 'thought-like' and less 'dreamlike'." An examination of their published data shows, however, that the critical feature of the drug nights was the absence of any dream reports scoring 6 or 7 on the fantasy scale. Thus the effect of the drug was to make dreams less real-at-the-time rather than to make them conceptual. Dreaming which is perceived to be 'unreal' or 'like a film' or 'only a dream' implies some degree of reflection on the part of the dreamer. This is 'secondary cognitive elaboration' in the terms of Molinari and Foulkes. It may be related to lack of active participation in the dream (all the active dreams in Carroll's study scored 6 or 7 on Foulkes' scale), but it is possible to be actively involved in a dream and yet realise it is 'only a/

/a dream'. This is perhaps one of the features which prevents all apparently frightening dreams from being felt as nightmarish. Thus Carroll's results are consistent with the view I shall adopt, that since phasic activity in REMPs is associated with the intrusion of bizarre material (Rechtschaffen and Watson have shown that phasic activity leads to bizarre mentation), then an absence of phasic activity allows secondary cognitive elaboration to predominate. The finding in these experiments that nitrazepam makes dreams less bizarre clearly ties in with this view. Perhaps a synthesis of Carroll's results and of these results can after all be achieved.

5. Alternative explanations of the lack of drug effects in these results:

This is a convenient point to consider whether there might be other reasons for the negative findings of these experiments. Was the choice of experimental nights poor? Maximal REM sleep rebound occurs on the first withdrawal night following amylobarbitone, but on the third following nitrazepam (Oswald and Priest, 1965). Did choice of the second withdrawal night lead to withdrawal effects being missed? Surely not, since EM profusion scores were high on the second withdrawal night. Further, a bad choice of dream collection nights cannot explain the lack of any effects of the active drugs, since initial drug nights produce greatest REM suppression (Oswald and Priest), and EM profusion was very much reduced on these nights.

Could there have been an effect of the drugs which was merely masked by high variability? The occurrence of a number of no-content reports might suggest this explanation. Changes in active/passive/

/passive dreaming and changes on the Foulkes' scale were however examined only in those reports with some content, and no effects were found even then. There was nothing in the initial inspection of the data which suggested that changes in the prevalence of activities or social interactions were present but were masked by high variance. And it should be remarked that if drug effects are so slight by comparison with other 'random' effects on dream content, this in itself is an important result, since drug effects on REM sleep are so dramatic.

Were the measures used simply not sensitive? The presence of differences as a function of time of night in the number of active dreams, in the Foulkes' fantasy scale ratings (Fig. 38), and in the number of strange or unfamiliar characters and the number of social interactions on the Hall and Van de Castle scales argue that all of these scales were sensitive enough to detect drug effects had they been present. Foulkes' scale has discriminated REM and sleep-onset reports in terms of the proportion of hallucinatory (real-at-the-time) experiences (Foulkes et al 1966); Carroll's study showed that active-passive ratings and the Foulkes' scale certainly did not suffer from lack of sensitivity. The correlation of active/passive dreaming with EM profusion both in this study and in those of Berger and Oswald (1962) and Dement and Wolpert (1958) shows that the former is a sensitive measure of the dream experience.

Could the EOG records have failed to pick up eye movements on drug nights because they were of smaller amplitude than on placebo/

/placebo nights? The recordings, with a gain of 100 microvolts per centimeter and time constant 0.1 sec should be capable of detecting eye movements of roughly 3 degrees amplitude or greater. Barbiturates and nitrazepam suppress smooth-tracking and convergence eye movements at doses comparable to those used here, but saccadic eye movements are virtually unaffected at these doses (Rashbass, 1961 ; Norris, 1970). Hence it is not possible to suppose that subjects had visually active dreams whose accompanying eye movements were merely reduced in amplitude to a point where they were not picked up by the recordings. Moreover, subjects' eye movements subsequent to awakening were clearly visible. The oculomotor effect of these drugs is present in waking, and it does not seem reasonable to suppose an effect selectively so much greater in REM sleep as to render small eye movements quite undetectable. No amplitude criterion was employed in scoring EMs in REMPs other than that they were visible on the EOG record. There was no visible suggestion of small but frequent EMs on drug nights. EMs in REMPs are of smaller amplitude under hypnotics (and larger amplitude on withdrawal - Figs. 16 and 17), but the amplitude of EMs on drug and on withdrawal differs by only a factor of two or three.

The lack of changes in dream reports from laboratory REMP wakings as a result of drug administration appears, then, to be 'genuine', not an artifact. What then of the very clear cut changes in dreaming reported by some of the subjects in their own estimates? Why were nightmares reported by two subjects on withdrawal when no other/

/other withdrawal effects were noted? I shall argue that increased anxiety on withdrawal interacts with the subjects' perception of their dreams, which is itself altered by the drugs. First I shall discuss the data from the 10cm lines, and then the nightmares.

6. The subjective estimates:

(a) Individual responses: Subjects' ratings of their own dreaming (Figs. 27, 28 and 29) show wide variations of response. Some subjects like the one illustrated in Fig. 27 showed the pattern of dreaming which had been predicted for all the subjects. Others, like subject J.S. (Fig. 29a) experienced a change at one point in the cycle of drug administration, but not in the fashion predicted. A "withdrawal effect" when this subject had experienced no depression of dreaming on the drug seems paradoxical. One subject reported very aggressive dreams on amylobarbitone (see Appendix IV) - quite the opposite of what had been expected. I have heard other anecdotal reports from people who have had very vivid dreams while taking sleeping tablets such as nitrazepam. Some of these dreams are quite clearly of hypnagogic origin, but others sound very much like REM dreams.

On the whole, the subjective changes reported in this study did not seem to be in any way correlated with individuals' dreams as collected on experimental nights. The subject who reported very aggressive dreams at home on amylobarbitone, however, did have laboratory dreams which were profuse in characters, activities/

/activities and aggressive interactions struck me at the time as reminiscent of the way some people become very aggressive after drinking substantial quantities of alcohol. Peoples' responses to alcohol are very varied - some are quietened down, others liven up; some become friendly, others aggressive. These responses can usually be related to the individual's personality. I would suggest that the individual's response to a sedative in his dreaming is probably related to his response to the sedative in terms of his waking behaviour.

If the response of dreams to drugs is a function of prior personality, it would seem hardly surprising that these drugs should have no consistent effect on dream content over a group of subjects. When this study was begun, it was not expected that personality would interact with the drugs. Although there are widespread personality correlates in dream recall (see review by Cohen, 1970, for example), it was anticipated that the drug effects would over-ride personality effects; though personality correlates are widespread, these correlates are not strong. Looking back, it is clearly unfortunate that the design of the study did not incorporate at least some personality dimension as a factor. This is certainly something which future research into drug effects on dreaming should do. The one study on the effects of drugs on dream content which did use a personality dimension as a factor (Deichsel, 1973) has already been referred to in Chapter 13. There was apparently an interaction between introversion-extraversion and the effect of the hypnotic on dreaming.

Extroverts' dreams were apparently affected to a greater degree, dreams having less aggression, anxiety, and less characters, becoming more like introverts' dreams without the hypnotic. Pivik and Foulkes (1966) noted an interaction of the effects of REM deprivation (by behavioural means) in the first half of the night with the personality dimension repression-sensitisation, when they examined dreams from REMPs in the second half of the same night.

In a rather more general frame of reference, Hauri and Van de Castle (1972) have invoked Lacey's concept of individual response specifically to explain contradictory findings on psychophysiological correlates of dreaming. They argue that differences between individual physiological responses to stress might explain reports of both positive and negative correlations between heart rate variability and dream intensity and emotionality. It seems possible that this argument might work in reverse: "individual response specificity" could explain why a drug whose effect can be categorised as 'sedative' can either increase or decrease 'intensity' of dreaming - or have no detectable effect - in a variety of subjects. As Witkin (1969), ^{and} Breger et al (1971) and others (1970) point out, personality interacts with the effect of a stressor in altering dream content.

(b) Vivid dreaming in withdrawal:

A striking feature of the subjective effects of the drugs is the increase in vivid dreaming on the first withdrawal day following amylobarbitone (Fig. 30). It will be recalled that in the second experiment subjects were asked to rate not only their 'dreaming'/'

/'dreaming' globally, but were asked to try and discriminate time spent dreaming from the quality of the dreaming (vivid, bizarre etc). All three ratings showed the same effect to a greater or lesser degree, so it may be that subjects were simply unable to discriminate these variables when at home. However, it may be that vividness of home dreaming is genuinely highly correlated with amount of dreaming. In this experiment time spent dreaming was hopefully held constant - at least subjects always got $7\frac{1}{2}$ minutes of REM before being woken. It is known from previous work that both of these drugs do depress the amount of the night spent in REM sleep. But in this experiment the 'intensity' of dreams was unaffected (note that the judge could not guess which dreams were drug dreams). What factors operating in the home might affect dreaming as recalled in the morning which were not operative in the laboratory conditions?

One obvious factor is that dreaming apparently changes as the REM sleep progresses. Foulkes (1966) describes how from longer REM sleep, more scenes with clear visual imagery are described, ratings for hostility, unpleasantness, dramatic quality, activity and emotion, including anxiety, are all raised. If morning recall of dreaming were derived from spontaneous wakings at the end of a REM sleep, then factors which affect time spent in REM sleep would ipso facto affect the quality of recalled dreams as well. But if home dream recall is also affected by dreams partially remembered from wakings during the night, further factors may operate. (Webb and Kersey, 1967, have shown that daytime dream recall can be accounted for solely in terms/

/terms of morning waking from REMPs for a sample from the man in the street - but subjects undergoing a dream experiment may become extra-conscious of their dreams and their REMPs). In particular, the probability of waking from a REMP during the night may be depressed by sedative drugs. If such wakings occur, the time available for rehearsal of the recalled dream fragments will be reduced, and so morning recollection will be lower (Portnoff et al, 1966). The content of material remembered may be biased by a tranquillising drug (an anxious dream may lose the anxiety in recollection). These factors might operate on identical dreams to make morning recall at home quite different on drug and placebo nights. These factors might explain why dreams at home were recalled as being more vivid and bizarre on withdrawal while there were no changes in the dreams got from REMP interruption.

7. The nightmare:

Why were two nightmares reported on drug withdrawal at home when almost no withdrawal effects were found in the laboratory collected dreams? Clearly the nightmares at home cannot be explained as being the result of a high profusion of EMs on withdrawal, for then we would have seen changes in the laboratory collected dreams as well. Moreover, Le Gassicke et al (1965) reported nightmares following withdrawal of tranlycypromine (Parnate, chemically related to amphetamine). Amphetamine itself does not appear to influence eye movement profusion (Baekeland, 1967) - at least there is no evidence of increases in EM profusion following withdrawal after regular use/

/use of amphetamine. Tranylcypromine resembles amphetamine in this respect (Oswald, personal communication), so the nightmares observed by Le Gassicke et al cannot be related to EM profusion.

An obvious factor which would seem relevant to the occurrence of nightmares at home is the fact that home dreams are on the whole 'spicier' than laboratory dreams. Specifically, both Domhoff and Kamiya (1964a) and Weisz and Foulkes (1970) have shown that home dreams contain more aggression than laboratory dreams. If nightmares are frightening on account of aggression by other characters, this could be relevant. But Weisz and Foulkes also found no significant difference in the rated unpleasantness of home and laboratory dreams. It might make sense that if in the laboratory withdrawal dreams were at least slightly different from baseline dreams, these differences might be accentuated at home. But the differences in home and laboratory reports would not seem capable of explaining the occurrence of nightmares at home, when there are no differences between baseline and withdrawal dreams in the laboratory.

As described in Chapter 12, nightmares have been frequently reported in the withdrawal of subjects dependent upon drugs. Could it be that the psychopathology of these subjects in some way interacts with drug withdrawal so as to produce nightmares? This seems highly unlikely, since the two subjects who had nightmares in these experiments, besides being screened for overt psychopathology, did not betray any suggestion of this during the course of the experiment. Oswald and Priest (1965), and Kales et al (1969b, 1971) have also/

/also noted nightmares in normal subjects in drug withdrawal.

Patients who cease taking sleeping pills at home frequently complain of "nightmares". It is possible that they do so at least partly because they expect that they will not sleep well when they stop their medication. We should consider the possibility that the two nightmares reported by subjects here were largely 'responses' resulting from the subjects being aware that the active drug had just been discontinued. How good was the double-blind in these experiments?

The objective of blind trials is to eliminate or at least reduce the possibility of effects being attributed to the active drug which were in fact the result of 'placebo effects' (Wolf, 1959¹) or similar phenomena. The essence of a single-blind trial is that the subject is given active drug and dummy in a form and under conditions which are always identical so that expectations on the part of the subject as to what the drug may do to him are evenly distributed throughout all experimental conditions.

A possible failure in this technique is that the experimenter may (quite unwittingly) give the subject hints as to whether or not he is on active drug. The subject need not necessarily be aware of the information being imparted to him: if the experimenter becomes very concerned at how the subject is feeling one day, then the/

¹Wolf S. The pharmacology of placebos. (1959) Pharmacol. Rev. 11 689-704.

/the experimenter may obtain the information he expects without any deliberate intention on his part, or awareness on the part of the subject. Because of this situation double-blind procedures attempt to keep both the subject and the experimenter unaware of the experimental condition the subject is in.

In these experiments, a double-blind procedure was run with all the amylobarbitone and placebo subjects. The E was, however, aware of the experimental design. Because he knew which nights would be drug and withdrawal nights, he was made unaware of which subject was on active drug. Any unintentional probing for symptoms would therefore have been directed equally to placebo and active drug subjects as long as E did not guess subjects' status. As was described on p.185, because the E saw the subjects' EEG, and heard the subjects discussing their sleep, the double-blind wore off as the experiment proceeded. Could this have 'produced' the nightmares reported by two subjects?

This is unlikely for two reasons. The first is that as has been already described (p.196) the interviews were carefully designed to minimise prompting by the E. The subjects were not questioned about their dreaming outside of the night-time interviews, except by being offered the 10 cm lines each day. They were not asked for any dream fragments that they could recall from their nights at home.

Thus it is arguable that E breaking the code should have had minimal effects on the reported dreams of the subjects. The fact that the E was predisposed towards obtaining positive results/

/results from the night-time wakening data, and in fact almost entirely negative results were obtained, argues that the precautions against experimenter bias must have operated effectively. In a study of experimenter effects on recall, Herman et al (1970) found the effects of both experimenter and subject bias to be non-significant on content derived from REMF interruption.

In the debriefing following the experiment, it was found that the subjects were in fact not capable of discriminating when they had been on drug (experienced, or trained subjects might be better able to do this). Almost all subjects who had actually been on placebo throughout were convinced that they had been on active drug at some point during the experiment. None of those actually on drug realised the design of placebo followed by drug, then back to placebo. One of the subjects who produced a nightmare was convinced it had nothing to do with the sleeping pills, but was a result of "other things" happening in his life.

Most subjects would not have known what changes to expect (not having encountered sleep research before). That the nightmares were not produced (as evidence of bad sleep, perhaps) when subjects became aware of a change in their pills is further suggested by the pattern of spontaneous reports. Spontaneous reports about dreaming at home came throughout the experiment, both from subjects on active drug and from those on placebo. They included spontaneous reports of very little dreaming, as well as reports of vivid dreams. The latter were not only reported/

/reported by subjects during the "withdrawal" period, further suggesting that unintended experimenter bias could not have been wholly responsible for the spontaneous reports by subjects.

For these reasons it does not seem reasonable to ascribe the two nightmares obtained to deficiencies in the experimental double-blind. Granted that the occurrence of nightmares on withdrawal was a "genuine" effect, and given from the discussion above that it was not a result of either high EM profusion, or the psychopathology of subjects, to what can this phenomenon be ascribed?

One of the features one would expect to occur at a time when nightmares occur is anxiety, since most nightmares¹ are presumably associated with high levels of anxiety. However, no changes in self-rated anxiety were found in this experiment. Nor were any changes in anxiety noted in the dreams collected in the laboratory, when those dreams were rated by an experienced judge. The possibility that the self-rating scale for anxiety is insufficiently sensitive seems unlikely, since self-rated anxiety was measured on a hundred-point scale (ten cm scale, measurements to nearest mm). Moreover, these scales have been shown to discriminate well in related situations (Aitken, 1969; Akindele et al, 1969).

Despite this lack of indication of increased anxiety on withdrawal, there remains at least a possibility that short-lived

¹The term nightmare is used to refer to the subject's experience. See footnote to page 229.

/short-lived increases in anxiety were overlooked. This possibility is suggested by the fact that O.O. Ogunremi, in recent observations in this laboratory as yet unpublished, did find increases in self-rated anxiety on withdrawal of amylobarbitone and a minor tranquilliser (he also found increases in night-time plasma cortisol). The reason why increases in anxiety could have been overlooked in this experiment is that all anxiety ratings were done in the evening. Although subjects were requested to give a rating for the whole day, it seems possible that evening levels of anxiety were reflected to a greater degree than morning levels. It has been pointed out that in a sense therefore every evening was a withdrawal evening. Kales et al (1969b) found increases in anxiety measured in the morning, with normal subjects on withdrawal from methyprylon and glutethimide. It seems therefore at least possible that anxiety was raised to a small degree on withdrawal from amylobarbitone, but that this was missed by the self-rating system used. Any such increase in anxiety would have had to be slight, or restricted entirely to the first withdrawal night, however, since increases in anxiety were not detected in the dreams collected in the lab. on the second withdrawal night. This suggests that besides anxiety, some other factor or factors must have been operating to produce the nightmares that were reported.

/One

One feature of the dreams collected in the night struck me. While listening to subjects telling their dream reports, I was interested that they would sometimes describe a scene full of guts and gore with apparent relish - certainly with no hint that they might have been revolted or horrified at the time. It is of course well known that normal dreams are frequently devoid of the emotions one would expect from the same circumstances in real life. Hall and Van de Castle (1966) have discussed this point in connection with the problems of scoring the presence of emotion in dreams. But it suggests that one feature of a nightmare is not only what happens to the dreamer, but how he perceives it. The nightmare is full of anxiety; it is also nasty, unpleasant, bad. Kales et al (1969b) found increases in dream unpleasantness following withdrawal of methyprylon and glutethimide in normal subjects at home. On a waking mood scale in their laboratory experiment they found a significant increase in depression following the drugs. Could it then be that subjects' dreams may be as active and aggressive on placebo, drug and withdrawal, but they perceive the dreams as worse, more frightening, more anxiety provoking, more depressing when they are suffering from hypnotic withdrawal? There are two other pieces of information which suggest that this is a contributory factor in causing subjects to regard some dreams as nightmares.

/Lewis

/Lewis (1969b) showed that subjects assessing their sleep (time to fall asleep, number of awakenings, total sleep time) overestimated how bad their sleep was after withdrawal from hypnotics. They think they slept worse than they really did. In a different context, the performance of athletes, Smith and Beecher (1960) found that athletes performed much better. Vice versa, when on amphetamine, they performed better but tended to regard their performance as worse than on placebo. Gottschalk et al (1960) also found an increase in the expression of feelings of positive emotional involvement, and feelings of bodily and emotional well-being in a study of the effect of perphenazine (a phenothiazine which reduces anxiety) on waking verbal material. The verbal material was a 5 minutes description of any interesting or dramatic personal experience.

Thus it seems very reasonable that our subjects on withdrawal from sedatives would have dreams which are normal in all but two respects. First, their dreams may contain more anxiety, at least on the first withdrawal night. This is particularly likely in dreams they have at home, where the longer REMPs on withdrawal will be associated with dreams higher in anxiety (see page 257); this will interact with any general tendency for anxiety to be increased in withdrawal. The tendency for home dreams in withdrawal to contain more anxiety, for the reasons just stated, will interact with subjects' altered perception to produce dream experiences which are perceived as especially nasty, depressing/

/depressing and anxiety provoking. If a dream contains a high level of anxiety, then the chances are high that it will be perceived as a nightmare.

A conclusion

Thus it would seem that the occurrence of nightmares on withdrawal from amylobarbitone is not inconsistent with the results of the rest of the experiment. With possible increases in generalised anxiety, a lengthening of REMPs at home on withdrawal, and altered perception, dreams which are in many respects like drug or placebo dreams in the laboratory can turn into nightmares at home.

Despite the absence of a noticeable increase in anxiety in this experiment on withdrawal, there is at least some evidence from other studies to suggest that measures of daytime and night-time anxiety in this experiment were not adequate enough to detect a temporary increase in anxiety lasting only a day or so. It is at least possible that in the conditions of this experiment (a moderate dose of tranquillising hypnotics for two weeks) an increase in anxiety on withdrawal might occur, but be slight enough to be detectable for no more than one day after withdrawal. (There is no reason to suppose that an anxiety 'rebound' need last as long as a REM rebound). If there were a brief increase in anxiety on the first withdrawal night, this would have been a contributory factor in producing nightmares at home then. But if it were only a brief effect, no increases in anxiety would have been noted in the dreams collected from the/

/the subjects on their second withdrawal night.

The important conclusion from this argument is that it is subjective factors, and emotional factors, which operate on waking thought processes as well as at night, that are responsible for the nightmares. The fact that nightmares occur at a time when the profusion of eye movements in REM sleep is especially high is thus a coincidence, and is not directly related to the nightmares. The positive findings of changes in our subjects' subjective estimates of their dreaming, and the two spontaneously reported nightmares are thus as important results as the largely negative findings of the rest of the experiment. One positive finding from the laboratory dreams, namely the effect of nitrazepam in potentiating everydayish dreams, also suggests a "tranquillising" effect which is likely to be operating on waking fantasy or non-directed 'day-dreaming' thought as well as on dream content. By contrast, the relation between EM profusion and dream content was marginal. I shall now develop this in some moresspeculative generalisations about the nature of dreaming.

CHAPTER NINETEEN

SOME SPECULATIVE CONCLUSIONS ABOUT DREAMING

1. Hypotheses

Dreaming was first associated with REM sleep by Aserinsky and Kleitman in 1953 , and the presence of eye movements just before the unfortunate subject was woken and described a visual experience led naturally to the idea that these eye movements might represent the dreamer looking about his dream world. The test of the scanning hypothesis by Roffwarg et al in 1962 seemed to confirm that this was in fact so, particularly since in the same year two sceptics were "pleased to report confirmation of ... a relation between profuse eye activity and an active dream fantasy" (Berger and Oswald, 1962). This seems to have been a turning point for the scanning hypothesis, however, for Foulkes' report that dreaming could occur in all stages of sleep appeared in the same year, and since then a lot of other evidence (discussed in Chapter 12) has accumulated which casts a great deal of doubt on the idea that eye movements in REM sleep have much relation to the dream content subsequently reported.

The scanning hypothesis has fared badly from Moskowitz and Berger's attempt (1969) to eliminate the bias inherent in Roffwarg's experiment. Certainly the scanning hypothesis receives no support from the results of the experiments described here. If REMPs containing almost no eye movements can yield dream reports which are indistinguishable from other reports when examined by a judge who expected the reports to be discriminable (as the judge initially did), then it seems/

/seems that the number of EMs cannot be related to the visual activity of the dreamer. It has been argued (Roffwarg, personal remarks) that maybe the scanning hypothesis is valid normally, but that the relationship between EMs and the dream is 'dissociated' or 'blocked' by various factors, such as the presence of psychoactive drugs in the system. Such a relation is then also blocked, presumably, by decortication (Jouvet, Pellin and Mounier, 1961), or indeed by the presence of NREM sleep. In the first case, visual imagery cannot be present, and in the second eye movements are not, but visual imagery may be. This postulate seems weak at the best, and I frankly cannot give it much credence.

If eye movements are not related to dream scanning, what of the hypothesis that EM profusion as an index of REM 'intensity' is related to an 'intensity' of the dream? The results of this experiment argue as strongly against this hypothesis as they do against the scanning hypothesis. Any such relation is very weak - correlations are low - and suggests that some third factor is influencing both the EM profusion and the dreams.

If some third factor is influencing both eye movements and also dreaming, what might it be? I have argued (Chapter 12, pp 140-144) that such a factor might be PGO spike activity. PGO activity is highly correlated with EM activity in the cat, and recent studies of mental correlates of 'phasic' events in both REM and NREM sleep suggest that PGO activity is associated with changes in ongoing dream content. Sudden changes of scene/

/scene or line of thought, and the abrupt appearance of new objects or people, may occur (Watson, 1972). (A suggestive analogy is perhaps the 'editing' of a film). Directly relevant to the possible association between active dreaming and PGO activity, Foulkes and Pope (1972) showed that phasic activity in REM sleep coincides with the cessation of thinking, watching and reflecting activities ('passive' dreaming).

Thus the marginal association between the 'intensity' of the REMP, and active dreaming, may be produced by PGO activity, correlated on the one hand with EM profusion scores, and on the other hand with the replacement of reflective (passive) dream content by active involvement.

Dream content 'intensity' is only marginally related to the 'intensity' of the REMP. What is the status of the 'quality' hypothesis? This hypothesis, as formulated by one of the independent judges, stated that any changes in dream content would be describable in terms of the effects of the drugs on the quality of experience, rather than on its intensity. Although the predicted effects on such items as the number of friendly interactions were not borne out, the positive findings of this study do suggest this sort of effect. Thus I have tried to show that the occurrence of withdrawal nightmares, and changes in dream content found by Kales et al (1969b) in their studies with hypnotic drugs, are instances of effects on the quality of thought processes.

I have/

I have tried to show (pp 261-263) that the changes in dream content found by Kales et al (1969b) and in this experiment are changes in the quality of thought processes which are not restricted to dreaming. Thus Kales et al found that waking depression increased when dream unpleasantness increased (they didn't actually rate dream depression or waking unpleasantness). The perception of sleep, performance and dreams are all affected in the same way by drug administration and withdrawal, whether the perception takes place by night (dreams) or by day (morning ratings of sleep, daytime ratings of performance). If the quality of daytime thought processes or perceptual processes is affected in the same way as the quality of night-time (dreaming) thought processes and perceptual processes, this fits into the idea of a continuity between waking and dreaming processes of perception and thought. Evidence on whether there exists a continuity or complementarity does not all favour continuity. For instance waking sexual desire is inversely correlated with sexuality in dreams, as Swanson and Foulkes showed in 1968). It would appear that complementarity prevails with aspects of 'drive': Hauri (1970) found that subjects dreamt less about activity following waking physical activity, and less about thinking and problem solving after studying. But in aspects of personality and mood, continuity is the rule (Foulkes, 1970; Kramer, 1970; Kramer et al, 1970). Thus dreams are most unpleasant during that phase of the menstrual cycle when waking depression is highest (Swanson and Foulkes, 1968), and MMPI/

/MMPI and behavioural ratings of hostility are correlated with the expression of hostility in dreams (Ben-Horin, 1967). Jung's idea (1960¹) that dreams have a function which is compensatory to waking thought processes might therefore be seen to have derived some support from recent studies. Jung distinguished his idea of compensation from that of mere complementation: he supposed that the attitudes expressed in dreams could be coincidental with, variations from, or opposite to the attitudes expressed in waking, according as the waking attitudes were 'adequate', or 'one-sided'. However, whether dream processes parallel waking thought-processes or complement them is seen by recent work to depend not on whether the waking attitudes are one-sided, but upon whether drive processes are involved or not.

We have noted that the hypnotics did not make REM mentation like NREM mentation in these experiments. Thus the characteristics of REM dreams are less a function of eye movement activity within the REM than a function of the presence of REM sleep per se. Maybe the quality of REM dreaming has more to do with a state of arousal than anything else. Hypotheses of this sort have been put forward by Zimmerman (1970) and by Rechtschaffen, Watson, Wincor and Molinari (1971).

Zimmerman's hypothesis (1970) is based on differences in mentation between 'light' and 'deep' sleepers (defined in terms of their auditory awakening thresholds). Light sleepers reported NREM experiences as 'dreaming' more often than did deep sleepers. Zimmerman proposed that 'heightened cerebral arousal, rather than specific physiological/

¹Jung, C.G. (1960). On the nature of dreams. In Read, H., Fordham, M., and Adler, G. (Eds.). The structure and dynamics of the psyche (The collected works of C.G. Jung, 8). Routledge, London, 281-297.

/physiological mechanisms, is the basic requirement for dreaming", or alternatively, that "dreaming is a function of cerebral arousal in the absence of reality contact". Notions of arousal are notoriously liable to be associated with over-simplification - the notion of one dimension of arousal is not consistent with the dissociation of various measures of arousal either in waking or in sleep. But the concept of cerebral arousal as judged by and restricted to the EEG can be meaningful. Thus few would argue with the statement that cerebral arousal is higher in alert wakefulness than in drowsiness (stage 1 sleep) or in REM sleep. Further, cerebral arousal is higher in drowsiness and REM sleep than in NREM stage 2, which contains more slow-wave activity, and NREM stage 4. Reports of 'thinking', of less organised mentation, tend to come from NREM sleep, and reports of 'dreaming', with a degree of organisation of mentation, from REM sleep and from stage 1 drowsiness (especially at sleep onset). Zimmerman's hypothesis therefore has a certain appeal. Zimmerman proposes that when NREM sleep is 'lighter', mentation becomes more dreamlike. This is quite consistent with the idea I am arguing, that dreaming is solely a function of heightened cerebral (EEG) arousal. For many people, dreaming will only occur in REM sleep, but in individuals whose NREM (stage 2) sleep is especially 'light', dreaming will also occur in NREM stage 2 sleep (dreaming is here contrasted with mere 'thinking').

The hypothesis put forward by Rechtschaffen, Watson, Wincor and Molinari (1971) related to brain stem arousal. They proposed that "tonic brain stem arousal, reflected in tonic extraocular activity,/"

/activity, may be associated with the emergence and/or recall of conscious experience". The status of a concept of brain stem arousal, particularly if it cannot be directly measured, seems weak. Nevertheless, Rechtschaffen et al have discriminated a tonic phenomenon which seems to originate in the brain stem, from related phasic phenomena, and shown that the tonic phenomena is associated with the degree of recall, and the phasic phenomena with the type of recall (intrusion of the bizarre).

2. A Synthesis

Dement's experiments on 'dream deprivation' (1960) led to the concept of a 'need to dream'. Such a view of dreaming has not been supported subsequently. The pharmacological evidence of a rebound/

/rebound in REM sleep exceeding that lost on drug, is simply not consistent with such a view, which also tied down dreaming to REM sleep. REM sleep is present in huge amounts at birth (Roffwarg et al 1966), and it appears that REM sleep may serve a function in the growth and maturation of the cortex at this time of life (Oswald, 1969c). REM sleep is also present in a wide variety of infra-human species in whom the 'need to dream' seems unlikely. Whether cats dream, or whether new-born babies dream seems to me an irrelevancy: they presumably have some sort of visual experience, however rudimentary, during REM sleep, and whether one considers such experience dreaming is a matter of personal preference. It would at least appear that new-born kittens are unlikely to have visual experiences in REM sleep, since their eyes have not opened. On the other hand experiments by Vaughan (1963), in which he trained monkeys to press a lever when they had visual experience, suggests that monkeys 'dream': they pressed the lever frantically during certain periods of sleep. Maybe cats have wish-fulfilment dreams when they twitch and chatter in REM sleep. What seems clear is that REM sleep takes place for a function² which is not psychological, but is more probably biochemical (Oswald, 1969c; West, 1969). However, other functions such as the maintenance of oculomotor control may also have attached themselves to REM sleep (Berger, 1970) - there is no reason to suppose REM sleep, any more than any other piece of behaviour, only serves one function. Despite the increasing body of evidence indicating a function for REM sleep which is either physiological or biochemical, there are still/

²The use of the term function is explained on pages 271-272.

/still attempts to formulate the function of REM sleep in terms of a functional theory of dreaming (e.g. Pearlman, 1970; Pearlman and Greenberg, 1970; Greenberg et al 1970). These theories have as their central theme the idea that dreaming functions to integrate new experience, to enable the organism to 'adapt' to new situations. Such theories merge into those which see a function for REM sleep in consolidating new learning, and 'reprogramming' (Newman and Evans, 1965; Dewan, 1969). At this point the idea that REM sleep serves to 'reprogram' the brain can be tied up with theories supposing an anabolic function of REM sleep in terms of brain recovery processes. The evidence for reprogramming, of the sort which might occur after brain damage, being associated with REM sleep is good (Greenberg and Dewan, 1969). On the other hand, the evidence that REM sleep is associated with learning processes of a less drastic kind is not good (e.g. Allen et al, 1972). The former approach ties in with the idea that REM sleep is essentially a biochemical phenomenon. To suppose a psychological function for dreaming in addition to an anabolic function, as Fisher (1967) does, seems unnecessary. It is more economical to assume that dreaming has no function.

I now wish to make the distinction between a piece of behaviour having a function, and a piece of behaviour being useful to a given individual. I am using the term function in a biological (rather than a medical) sense. Thus a piece of behaviour may be said to serve a function if it leads to consequences which are necessary for the survival of the species. Thus behaviour has a biochemical function if it/

/it leads to biochemical consequences necessary for survival ,
etc. If on the other hand a piece of behaviour leads to consequences
which aid the survival of the individual, but which are not necessary
for the survival of all individuals of the species, then that behaviour
may be said to be useful for the individual.

Thus the statement that dreaming as a mental process (i.e. as
distinct from REM sleep) serves no (psychological) function for the
species does not imply that dreaming need always be useless for any
individual. In an evolutionary sense, I would argue that dreaming
performs no function. It may, however, be a useful by-product for
an individual in a state of stress, for example.

It would appear that thought and dream processes go on in sleep to a
large degree independent of the physiology of sleep. As Hall (1967) has
put it "now it is known that dreaming can and does occur during any stage
of sleep, it follows that all of the distinctive neurophysiological
concomitants of REM sleep have little or nothing to do with dreaming or
dreams". The one constraining influence on the type of mentation is the
degree of cerebral arousal. Humans do not live in order to develop
scientific theories, but such activity may be a useful by-product of our
waking life. In similar fashion dreaming may be a useful by-product of
a brain which is conscious for long periods every night, with no immediate
problems (like finding the next meal) to solve.

REM sleep is on the whole associated with dreaming. Because our recall
for the type of mentation that occurs in NREM sleep is low, and/or because
our recall for events is low in NREM sleep, the amount of dreaming most of
us feel we experience is roughly correlated with the amount of REM sleep
we get. But dreaming does not imply/

/imply the occurrence of REM sleep: Vogel et al (1971) demonstrated clearly by getting judges to try and classify reports that "REM sleep does not contain a unique mentation". There are changes in the type of mental activity we experience during the night, but mental activity of some form or another goes on all, or almost all of the night. To a considerable degree there is a continuity in our mental processes throughout the day and night. As Calvin Hall puts it in his book (1966) "the images of our dreams are pictures of our conceptions": our dreams reflect the way we perceive the world.

I should like to argue that dreaming can be considered as a form of thinking which goes on when we are not capable of thinking abstractly. Under a state of reduced awareness and alertness, our 'thinking' largely takes the form of visual images, or visual metaphors and symbols. This thinking can sometimes be useful. An analogy which I wish to suggest is that of a man waiting on a platform for a train. He is sitting on a bench. His "function" in being there is to catch a train, for some reason which is not our immediate concern. But while he is there he is thinking. No more than the sleeping person can he stop thinking. His thoughts will probably roam, they will be disorganised and on a somewhat loose level, especially if his alertness and vigilance is lowered either because he is tired, or sleepy, or has had a drink. His thought will probably be of a disorganised, drifting, non-directed kind. He may conjure up various images. If he has nothing on his mind, his thoughts and images will not have any particular/

/particular significance, and they might seem as strange and illogical as many of our dreams. But if he has something 'on his mind' his thoughts will in all probability revolve in a non-directed fashion around his problems - his wife, or his work. Such thoughts may be part of his adapting to his problems. He may come to admit in a rather undefined way some aspects of himself he would deny in a more alert, controlled frame of mind. In similar fashion one's thought processes and dreams in sleep may help to adapt to one's problems; in a state where one's waking forms of defence (whether "repressive" or "intellectualising") are reduced, one may admit things one would deny when alert.

Most night-time mental activity will be as disorganised and useless as that part of our waking thought when we 'drift off' for a few moments. It is to my mind as absurd in this conception to regard dreaming as having a function as it would be to suppose our day-dreaming thoughts had a 'function'.

It is perhaps appropriate at this point to make clear that these ideas have little relation to those of Freud (1932) on the nature of dreams. In the first section of this chapter I described why Jung's ideas¹ on the nature of the dream have been superceded by recent work. The ideas I am putting forward here have more in common with those of Eysenck (1957b, 1972)³ and with those of Hall (1966). Thus I am in agreement with Hall when he states (p.14,17) that "Dreams also yield information about the dreamer's conception of the world. This world outside may be viewed as benign, hostile, turbulent, sorrowful,/"

³Eysenck, H.J. (1957b). The interpretation of dreams. In: Sense and Nonsense in psychology. Penguin, Harmondsworth, 142-174.

Eysenck, H.J. (1972) The experimental study of Freudian concepts. Bull. Brit. Psychol. Soc. 25, 261-267.

/sorrowful, lonely, degraded and in numerous other ways

Dreams, however, have a way of cutting through the pretensions and delusions of waking life and bringing the dreamer face to face with his real problems". On the other hand, I do not see reason to accept Hall's ideas that unpleasant, terrifying dreams, and nightmares, are punishment dreams. I have already argued in Chapter 18 that there are other reasons why people have nightmares. One does not necessarily have nightmares because one "has violated one of the commandments of his conscience. He has rebelled against authority, or he has gratified a forbidden wish, or he has committed a misdeed. The nightmare is the price he pays for doing wrong". (Hall, 1966, p.16).

Eysenck's viewpoint, stated at some length in Eysenck (1957b³), may be briefly summarised thus: "the mind in sleep does not cease to be active ... this activity ... continues to be concerned with problems of everyday life; the dream being a more primitive form of mental activity than waking thought, this dream activity expresses itself in pictorial and symbolic form. The function of symbolism is not to avoid the censor; it serves an adjectival function ..." (Eysenck, 1957b³). When Eysenck refers to dreaming as being more "primitive", he appears to mean that in sleep mental activity is 'at a lower level'.

Clearly these viewpoints, and the one espoused here, are radically different from that of Freud (1932). First, Freud holds to the distinction between manifest and latent dream content. Throughout the description and discussion of these experiments, I have referred exclusively to manifest dream content (excepting in the discussion of the psychodynamically oriented scales of Whitman et al 1961). I/

/I have concerned myself with the thought processes of the person when he is awake, or asleep. The concept of a latent content is unnecessary to the discussion I have already given in this chapter. As Hall (1966, p. xviii) has put it "As a matter of fact, it could be said that there is no such thing as the latent content of a dream. A dream is a manifest experience, and what is latent lies outside the dream and in the verbal material that the dreamer reports when he is asked to free associate".

The second of Freud's major hypotheses was that all dreams are wishfulfilments in some sense: "The dream is the (disguised) fulfilment of a (suppressed, repressed) wish". (1932, p. 164). While much of our daytime (and night-time) thinking may be in the form of wish fulfilments, I do not propose that all dreams fulfil a wish in some sense. Freud's conception of the function of distortions in dreams also finds no support here: there is no evidence in the work reviewed for his conception that "the censorship ... practises dream-distortion ... in order to prevent the development of anxiety or other forms of painful affect". (1932, p.260).

Freud proposed four mechanisms of 'dream-work': condensation, displacement, symbolism and secondary elaboration. In the viewpoint from which I am arguing, there is no reason to suppose that condensation does not occur - in waking as well as in sleep, a symbol can represent more than one idea. This point was discussed in Chapter 15; essentially it is plausible that to a given individual shooting a cow could symbolise both aggressive and sexual feelings towards his mother.

If the concept of a latent dream content is not used (and I do not see the need for this theoretical construct), then clearly Freud's/

/Freud's concept of displacement must also be abandoned, since it has no meaning unless a distinction is drawn between manifest and latent contents - displacement occurring between them.

I have already made it clear that Hall's (1966) view of symbolism is much more parsimonious than Freud's conception of symbolism as essentially a disguise. Both Hall and Eysenck (1957) have argued this point more eloquently than I could do here.

That secondary elaboration takes place between the dream experience and the telling of it few would deny. "The result of its efforts is that the dream loses the appearance of absurdity and incoherence, and approaches the pattern of an intelligible experience" (Freud, 1932, p.453). What is at issue is whether this process has a function, and if it does, whether that function is to disguise (as Freud maintains), or whether it merely serves to make the experience more comprehensible to our rational waking minds. It might also seem plausible that much of this process is in fact secondary to forgetting: pieces of experience which cannot be 'fitted in' to the rest may be more easily forgotten.

Thus it is not the existence of three of Freud's four processes of dream work, condensation, symbolism and secondary elaboration, which is at issue, but their function. In contrast to Freud's view of an active process of disguise, the view advanced here has more in common with one of the theories Freud reviewed (and rejected) in the introductory chapter of *The Interpretation of Dreams*. Many of these theories contain the germs of the various positions prevalent today; the one closest to the present position is that of Delage (1891, quoted by/

/by Freud): "En somme le rêve est le produit de la pensée errante,....selon que l'activité actuelle du cerveau est plus ou moins abolie par le sommeil."

Thus while Freud (1966, p.278⁴) supposes that the incongruities and absurdities of dream content arise by way of external associations and unconscious thoughts, the argument that I have developed is that these incongruities are more often the result of PGO activity intruding into the ongoing mental processes of the sleeping cortex.

In conclusion I would thus propose that we see REM dreaming as that part of ongoing thought processes which takes place when the level of cerebral arousal is sufficient to sustain some degree of organisation of mental processes. The phasic events of REM sleep, bursts of eye movements, saw-tooth waves, periorbital EMG activity all seem to be the result of activity in the brain stem. Watson (1972) has shown that PIPs (correlated with PGO activity) are associated with the occurrence of bizarre elements in REM dreams. It seems reasonable to conclude that it is the PGO spike activity which is responsible for the appearance of the bizarre elements. Thus the dreamer might be supposed to be "distracted" from his "line of thought" by the bursts of activity intruding into the cortex from below. His dream becomes bizarre as objects or people suddenly appear from nowhere. He ceases thinking, /

⁴Freud, S. (1966) The psychopathology of everyday life.
Transl. Tyson, A. Benn, London.

/thinking, talking about or explaining what is happening to him (Molinari and Foulkes' Secondary cognitive elaboration), and instead becomes involved in the events surrounding him. His whole attention is taken by the intrusion of unexpected events, so that he has no time to think about them; he is in fact actively involved, as Dement and Wolpert described in 1958.

3. A few comments as reflections on the omissions of this study

Given the approach to dreaming on which I have speculated, and the opportunities missed in these experiments, there are various suggestions which can be made for future work in this field. These can be divided into suggestions about methodology, and suggestions as to ways of testing some of the hypotheses discussed.

First, if at all practicable, experiments should clearly be completely double-blind. With only one experimenter working nights this is not possible, as he will see the EEG before interviewing the subjects. What is therefore needed is two experimenters, one of whom does not see the EEG, and only interviews the subjects. Ideally, a third experimenter who is unfamiliar with the hypotheses should do all the scoring.

Secondly, more information can be gained if two types of REM waking are made: phasic and tonic awakenings. Now that Rechtschaffen and co-workers have developed techniques for recording PIPs (1971, 1972), it would seem preferable to use these as indices of phasic activity where the influence of FGO/

/PGO activity on dream content is the objective of interest.

Thirdly, if it is supposed that there is continuity between waking and sleeping modes of mentation, then studies of drug effects should include personality dimensions as one variable. In particular, it is hypothesised from Eysenck's work (1963, 1967) that the effects of sedative drugs on dream content should be greater with extraverts than with introverts: an interaction effect is predicted.

Studies such as those of Bussell et al (1972), where waking conditions are included as one of the control conditions (in this case a study of eye movements and visual imagery) are also indicated if it is desired to test whether waking and sleeping thought processes are in fact parallel. In drug studies the procedure of Gottschalk et al (1960), where the subject is asked to produce a verbal report (e.g. a story), may be used to provide a waking condition. Such verbal reports can be analysed by the same procedures of content analysis as dream reports. It seems a pity that the analogy between certain waking thought processes and NREM mental processes suggested by Rechtschaffen et al in 1963 has not been followed up.

As regards methods of content analysis, it would seem from these experiments that in studies of dreaming which are not concerned with normative data or with differences between populations (sex, age, crosscultural studies for example), it is/

/is more efficient and productive to use rating scales, rather than content analysis using elements within the dream as a basic unit.

There are various issues raised by these results which suggest the need for further tests of the hypotheses I have advanced. The first issue is whether anxiety is indeed raised on withdrawal from such sedatives and tranquillisers as were used in this study. This can be better treated by obtaining morning ratings of anxiety, and by sampling blood cortisol levels during sleep. To test the mechanisms I have suggested as being responsible for the occurrence of nightmares in drug withdrawal, what is needed is studies comparing baseline, and withdrawal conditions after higher doses and longer administration periods, with length of pre-awakening REMP as an additional factor. Since it was hypothesised that it is chiefly longer REMPs on withdrawal which allow the development of high dream anxiety, an interaction would be expected between withdrawal and long REMP length. The use of REMPs exclusively from the end of the night would allow such a test to be made. This would also allow replication of Foulkes' original result (1966) that longer REMPs are associated with higher anxiety in the dream content.

The 'quality' hypothesis specifically can be investigated by analysis of verbal (daytime) ~~verbal~~ reports under tranquillising, sedative, and stimulant drugs, generating specific hypotheses from/

/from the results of such analysis, and testing whether the same changes occur in REM dream reports as occur on waking fantasy material.

The continuity hypothesis is now in need of a general test to see whether it is indeed the case that complementarity holds for a variety of drive situations, and continuity holds for non-drive situations (mood, hedonic tone, for instance).

Whether PGO activity is actually responsible for the occurrence of bizarre, out-of-context events in dreams could be tested by comparing phasic and tonic REM reports with sleep-onset reports. It is predicted that if reports of the very last experience are examined, tonic REM reports should not be discriminable from sleep onset reports, whereas phasic REM reports will be readily discriminable. So far tests of whether REM and sleep onset reports are discriminable have not distinguished phasic from tonic REM awakenings.

The hypothesis that dreaming (as opposed to thinking) is dependent on the level of cerebral (EEG) arousal can be tested by a comparison of the effects of placebo with amphetamine and tryptophan. Amphetamine is a stimulant, but does not apparently affect the profusion of eye movements. It would therefore be predicted (as Zimmerman, 1970, suggested) that amphetamine would make content from REM wakings less thought-like than a control condition; dream content would be expected/

/expected to be more active. By contrast, tryptophan has been reported to increase eye movement profusion (see Chapter 11), but it is predicted on the present hypothesis that its effect on dream content as compared with placebo would be none, apart from a possible effect in reducing depressive elements (tryptophan is in use as an antidepressant).

The use of both phasic and tonic awakenings should help to clarify the relation of dream content to PGO activity occurring in REM sleep as distinct from the tonic EEG arousal which characterises REM sleep. Thus it would be predicted that in an experiment of the present sort, tranquillisers would not reduce the bizarreness of reports of the very last experience derived from phasic awakenings, if PGO activity is responsible for this. However, to the extent to which the drug tranquillises all thought processes, it is predicted that REM-tonic awakenings would still produce reports which were more everydayish than control reports.

It is to be hoped that the above suggestions will allow the hypotheses I advanced in these last two chapters to be put to the test.

I don't have no one to cheat
Don't have no one to beat
You know I just need some room to uncurl
I don't have no aim in view
Just some dreams to pursue
As I wallow around in the world

Good as Gone

Robin Williamson

The Incredible String Band

APPENDIX I.

A summary of the scales and scoring symbols for Characters, Activities, Social interactions and Emotions.

(From Hall and Van de Castle, 1966).

CHARACTERS:

(a) Number:

1. An individual
 2. A group
 3. An individual
 4. A group
 5. An individual
 6. A group
 7. The original form of a character who undergoes metamorphosis.
 8. The final form of a character who undergoes metamorphosis.
- } Dead characters
- } Imaginary, fictional or dramatic characters

(b) Sex:

- M Male
F Female
I Indeterminate
J Joint (e.g. a crowd)

(c) Identity:

(i) Familiar characters:

- F Father of the dreamer
M Mother " "
X Parents " "
B Brother " "
T Sister " "
H Husband " "
W Wife " "
A Son " "
D Daughter " "
C Child " "
I Infant " "
Y Family member (indeterminate)
R Relative of the dreamer
K Known to the dreamer in real life, but not family member or relative
P Prominent person

(ii) Unfamiliar characters:

- O Character identified by occupation only
E Character identified by ethnic origins only
S Stranger
U Identity uncertain, includes characters known to the dreamer in the dream but not identifiable in real life.

(d) Age:

A Adult
T Teenager
C Child
B Baby

(e) Miscellaneous:

ANI Ainal
CZZ Creature

ACTIVITIES

- P Physical: voluntary movement of the whole or part of the body while the character remains more or less in one place.
- M Movement: change of physical location by self-propelled movement of the body.
- L Location change: movement in a spatial dimension to a different location by any means other than self-propelled muscular activity. There must be indication that the new surroundings appeared after intervening travel, rather than an abrupt shift in setting.
- V Verbal: any type of vocalisation.
- E Expressive communication: non-verbal activities such as laughing, crying, smiling, scowling, gasping.
- S Visual: seeing, noticing, reading, peeking, glancing, inspecting and similar activities.
- A Auditory: any type of hearing or listening behaviour.
- C Thinking: deliberate, continued mental effort possessing a goal directed or problem solving quality. Brief, transient mental activity is not scored.

SOCIAL INTERACTIONS

(a) Aggressive interactions:

Physical:

- A 8 An aggressive act which results in the death of a character.
- A 7 An aggressive act which involves an attempt to physically harm a character.
- A 6 An aggressive act which involves a character being chased, captured, confined, or physically coerced.
- A 5 An aggressive act which involves the theft or destruction of possessions belonging to a character.

Verbal or covert:

- A 4 An aggressive act in which a serious accusation or verbal threat of harm is made against a character.
- A 3 Attempts to reject, exploit, control or verbally coerce a character. Any type of negativistic behaviour.
- A 2 Aggression displayed through verbal or expressive activity.
- A 1 Covert feeling of hostility or anger without any overt expression of aggression.

(b) Friendly interactions:

- F 7 Desire for a long term close relationship: getting married, becoming engaged, falling in love.
- F 6 Socially acceptable forms of physical contact: shaking hands, cuddling a baby, dancing, kissing and embracing are included when clearly non-sexual.
- F 5 Taking the initiative in sharing a pleasant social activity: visiting, asking for a date. Simply participating jointly in an activity is not scored.
- F 4 Extending assistance, offering to do so, helping, protecting and rescuing.
- F 3 Offering a gift or loaning a possession.
- F 2 Verbal or gestural expressions of friendliness: smiling, greeting, 'phoning or writing with a friendly purpose, sympathising and praising
- F 1 Friendliness is felt but not expressed overtly.

(c) Sexual interactions:

S 5 A character has or attempts to have sexual intercourse with another character.

S 4 Various types of fore-play activities, fondling and petting.

S 3 Necking and non-platonic kissing.

S 2 Sexual overtures or propositions.

S 1 Sexual thoughts or fantasies.

EMOTIONS

AN Anger, annoyance, irritation, infuriation.

AP Apprehension, fear, anxiety, guilt, embarrassment.

HA Happiness, contentment, amusement, joy, exhilaration, relaxation.

SD Sadness (not physical distress), disappointment, depression.

CO Confusion, surprise, amazement, doubt, indecision, puzzlement.

Emotions are only scored when they are actually described, or when accompanying autonomic activity is described ('tears were running down her face'). They are not scored on the basis of inference: e.g. the dreamer being in a torture chamber is no ground for scoring fear.

APPENDIX II.

Hostility, Dependency, Anxiety, Motility, Homosexuality, Heterosexuality and Intimacy.

(From Whitman et al 1961).

Each phrase is scored separately on each of the following scales. For each scale, the scores of all phrases are then added; this total ("raw") score may then be corrected for the length of the report.

HOSTILITY

Rating

Scoring from
Most to Least

6. Human: Death or death threat by stabbing, shooting, pushing, striking, hit by car, drowning, illness, warfare, animal attack, mutilation, drive to insanity, violence.
5. Non Human: Equivalent destruction of animate or inanimate objects other than human.
4. Human: Injury or injury threat by fight, accident, illness, abandonment, rendered helpless, robbed.
3. Non Human: Equivalent injury of animate or inanimate objects other than human.
2. Human: Discomfort or discomfort threat by minor difficulty, hurt, annoyance, failure, inappropriate behaviour.
1. Non Human: Equivalent discomfort of animate or inanimate objects other than human.

DEPENDENCY

Scoring from
Most to Least

6. Total reliance on an object or institution (including specific references to eating or food).
5. Total reliance on a group.
4. Total reliance on an individual.
3. Partial reliance on an object or institution.
2. Partial reliance on a group.
1. Partial reliance on an individual.

ANXIETY

Rating

Scoring
from most
to least

6. Most extreme ever reported (Panic).
5. Extremely intense (Dread).
4. Unusually intense (Danger).
3. Intense (Foreboding).
2. Impressive (Apprehension).
1. Distinct but not impressive (Unpleasant anticipation).

MOTILITY

Rating

Scoring
from most
to least

6. Accelerated animate motor activity (springing, racing, sexual intercourse, etc.)
5. Vigorous animate activity (walking, rapidly, running, jumping etc).
4. Moderate animate activity (walking, driving a car, going some place by own motor power etc).
3. Minimal animate activity (talking, watching, eating etc).
2. Vigorous inanimate activity (rapidly growing things, cars moving, boats racing etc).
1. Minimal inanimate activity (light winds etc).

HOMOSEXUALITY

(Substitute woman for man with female subjects)

Rating

Scoring
from most
to least

6. Direct sexual expression with a man.
5. A man being alone with another man in situation with overt sexual possibilities or overtones (sleeping, lying down, brushing together etc).
4. Involved interchange between men including manipulation, joking, teasing or intimate conversation.
3. The appearance of men (two or more) with active interchange.
2. The appearance of two men but without active interchange.
1. The appearance of more than two men but without active interchange. (Minus 2 from each score if the male or female in the dream is a child).

HETEROSEXUALITY

(Substitute man for woman with female subjects).

Scoring
from most to
least

Rating

6. Direct sexual expression with a woman (coitus). (Including symbolic references).
5. Sex of foreplay type with a woman (including symbolic references).
4. Dating or being alone with a woman and/or in a situation with sexual possibilities.
3. Involved interchange with a woman including posing and conversation.
2. The appearance of one or more women in which he is the only male.
1. The appearance of one or more women in the dream but one or more other males are present. (Minus 2 from each score if the male or female in the dream is a child).

INTIMACY

Rating

6. A person is with people in an intimate relation with them (including touching and sexuality).
5. A person is with people with close interaction with them (talking, working together, common tasks etc).
4. A person is with animals in close interaction.
3. A person is with people at a distance but with interaction with them or close with animals without interaction.
2. A person is with people at a distance without interaction with them (visual, etc).
1. A person is alone in a dream without other people, or with living things but without interaction with them.

APPENDIX III

The Dreamlike Fantasy (Df) Scale:

(Foulkes, Spear and Symonds, 1966).

The scale contains 8 points (listed in Foulkes et al 1966, as extending 1 to 8, but usually assigned values from 0 to 7):

0	--	No recall	Feels mind was blank.
1	--	No recall	Feels he was experiencing something, but forgets what.
2	---	Recall	Conceptual (no sensory imagery), everydayish content.
3	--	Recall	Conceptual, bizarre content.
4	--	Recall	Perceptual (sensory imagery), non-hallucinatory (didn't believe experience was real), everydayish content.
5	--	Recall	Perceptual, non-hallucinatory, bizarre content.
6	--	Recall	Perceptual, hallucinatory (believed events he imagined were really happening), everydayish content.
7	--	Recall	Perceptual, hallucinatory, bizarre content.

"Recall" is defined as the mention of at least one item of specific content (not "I was thinking" but "I was thinking about home"; not "I saw images" or "I had feelings" but "I saw a car" or "I felt sad"; etc.).

APPENDIX IV

Dreams reported by subjects at home during the study.

- (1) A self-reported 'nightmare' dreamt by a subject in Experiment 1, on his first withdrawal night following 400mg amylobarbitone:

"We were all in a barn which was next to the sea and which was very dark and damp. We'd just found it as a place to stay for the night and I felt as though I was on LSD or something. Everything seemed disorganised and to be happening with no sort of control by me. The I was queueing for a bus just outside the barn with one of those who (had been inside) with me. It was as though a party had ended or something similar and we had to get home. There was someone else waiting for a bus at that stop and we were both worried about him seeing we had all these pills with us. Also I thought my friend was really ill and I was worried, frantic about myself too as though I'd been overdosed or something. The bus didn't come and we waited a long time. Then I was back in the barn. Perhaps I had to run back into it but the police had come and everyone in the barn knew things had gone wrong but didn't know what to do.

- (11) Another self-reported 'nightmare' dreamt by a subject in Experiment 2, under the same conditions. His 'first since childhood'.

"The dream started off with my mind seeing a model of small creature with horns and wings (face similar to that of the devil) being put into a bird cage. This gruesome creature kept appearing throughout the dream as a very evil being, and in the back of my mind I knew it had to be destroyed. At the second stage the being grew and grew until it broke the cage apart and broke loose. The next thing I knew I was back to the same setting watching this horrible little devil trying to break out of the cage, gnawing at the bars. Suddenly it started to grow again. Like a flash my hand was inside the cage grabbing at its throat but it transformed itself into a bedgie and I held it by the body and started hitting its head against a table. I kept on beating and beating.

- (iii) A "very vivid, very aggressive dream" reported from home by a subject in Experiment 1, while on 400mg amylobarbitone.

"The dream as such was extremely aggressive. I and Colin were both on horseback in Paddington Station. The platforms were crowded with people, there was great panic and confusion, trains letting off steam, people shouting. Completely frenetic. We were dashing up and down the platform on horses, lashing the people into the varriages, really smashing them hard, they were shouting and screaming. We galloped up and down the carriages riding the horses into the corwd. The noise of the horses ' shoes on the platform was tremendous, echoing over the station.

APPENDIX V

Some Examples of Laboratory Dream Reports

(a) Dreams predicted and dreams obtained

- (1) An example of the sort of report we expected to collect on active drug nights - thoughtlike, with little action:

529

"Could you tell me what was passing through your mind?"

"It was something to do with pinching something ... there was a crowd of us and we needed (?) something that was in this pile. It wasn't ours, it belonged to another friend. We were discussing whether to pinch it for a little while or not ..."

"Could you possibly repeat that more clearly?"

"Yes, it was a crowd of us discussing whether or not to pinch or take for a loan of this scooter sort of thing ... we didn't decide or anything before you woke me up ... that's all I can get just now, all I can remember."

650

- (11) An example of the sort of report we expected to hear on withdrawal nights - vivid, very visual, a variety of action, long and involved:

"Okay, there was in particular, there was, I was back in the, I was in Buccleugh Street, where in front of James Thins Paperback Bookshop and it was very dark it was at night, and it had just been raining, and the roads were sort of wet and glistening, just at the junction between Buccleugh Street and Buccleugh Place and I was standing with my back against the sort of windowpane of James Thin, and I saw these three cars coming, two from one direction and one from the other and one was coming very ... downhill fast from sort of the Appleton Tower direction, and the other two were coming up the way past the Meadow just as the two were coming past the Meadow one started to overtake the other and there was a fantastic pile-up, no car actually hit the other dead on, but sort of they all sort of started (?) weaving in and out, they all managed to end up in some sort of weird lozenge shape because there were two other cars arrived on the scene almost ... they miraculously appeared out of thin air, and they were sort of involved in the pile-up as well. But the ... none was really hurt badly but what was really impressive was the way that the cars sort of concertina-ed together, you know, sort of in and out, sort of geometric fashion sped down the road trying to avoid each other, and they all became interlocked and came to a standstill. There was a great crash and people got out, with blood pouring no one was sort of mortally injured or anything. no one dead. people were just cut with bruises and I

immediately sort of came over from Thin's Bookshop and sort of helped this old guy who was completely incoherent and was pouring blood ... that was one thing that went through my mind since the first time, you woke me, and, and the amazing thing about it was the clarity of the whole, of the whole sort of dream, in terms of vision. Everything was really incredibly clear everything was exaggerated, almost in fact as if it was some mirror of some kind, accentuated lights, lights of different kinds, it was amazingly clear, then it sort of progressed to talking with the other people, I'm not quite sure whether they were, whether I knew these people, but we were talking about lots of little things, talking about collecting tickets, collecting tickets and cleaning cars. Plus also started sort of getting ... I knew that ... I've woken once or twice since, I think, since you first wakened me, and hence I've been getting a series of different dreams, I think. There ... the next thing I was thinking about was sort of, was showing, simply showing people over the city of Edinburgh, you know, sort of driving around in a car showing these foreign people around and in particular we went on a drive out to Cramond Island. Pretty prosaic, just sort of going down there ... and somehow getting lost on the way, and taking hours to reach the place it was very simple we could always see, we could always see Cramond Island in the distance, but we could never get there. Which is really strange because you can't see Cramond Island at all until you're actually almost up against it. We could always see Cramond Island in the distance its outlines no matter how if you made a straight bee-line for it, it always seemed to sort of remain the ... sort of, you know, a constant distance from where we want. It never seemed to get any closer. It was really sort of frustrating. I don't think I can remember very much more, Hugh, actually, no."

- (iii) An example of a dream report collected from a drug night (200mg amylobarbitone) which did not conform to our predictions. I.C. guessed that the dream was collected on a withdrawal night. He rated it as an "active" report.

506

"Could you tell me what was passing through your mind please"

"There was a fire and somebody ... in a house, and somebody was jumping, I don't know where he was jumping from or to, jumping and he over, no, he jumped something to do with this fire; there was a window; through the window ... either from it or to it ... and it was in the ... top storey of a house ... with the standard triangular window ..."

"Go on."

"... I can't remember anything before that ... no ..."

"Anymore about this then, please?"

"...Things happened very very quickly ... it's as though, when

you asked me, when I, when I woke up, I could remember for a second the ... remember the thing in a time-sequence; there was just the second of a fire being in the top of some building with flames going like ... like ... like mad, and then the jump, and there was the feeling of everything was very very urgent ... that I had to jump out, ... yes, it was me doing the jumping out incidentally ..."

"Go on."

"... The place was probably Fountain hall Road except for the shape of the window, but I get the feeling it could be, but I don't get the feeling it is, ... but more and more I've a feeling it probably was; I don't know why, probably because it's the only well, it's the only place I can, probably really fit to that immediately. It is, was, then, I was jumping out into the front garden and there weren't any trees ..."

"Any more details?"

"The window was open, thank goodness ... or at least, I didn't go through any glass ... but I think it was closed, now; It was closed and I didn't go through any glass ..."

"Anything else?"

"... I'm even more sure now that it was Fountain hall Road ..."

"Anything earlier, anything before this?"

"... Nothing I can remember; nothing I can even feel that I had remembered and had forgotten ... the first thing I can remember about being awake was just this, and nothing more ... I even remembered, I could remember this before it, it seemed so urgent that I remembered this quite before I saw you, heard you, sort of."

"Any more details about this?"

"... I was still vertical although curled up a little, when I jumped through the window, I didn't dive through; I don't know if that's any good! I think that's about, practically, it must be complete detail, very nearly, yes."

"O,K, Were you thinking or dreaming, would you say?"

"Dreaming ... and yes, participating, very much so!"

"Anything else at all?"

"No."

"Fine, thanks, goodnight."

- (iv) An example of a dream report which both judges (I.O. and H.F.) guessed as being a 'drug' report, which was in fact derived from the second withdrawal night following 400 mg amylobarbitone:

566

"I dreamt that I was on a road check ... because in this area there property level had risen really high and the people were starving ... they were wanting to get potatoes so that the whole thing had escalated that this family were trying to protect the rest of the families in the area by getting potatoes for them and they were having ... the last ... the thing I was thinking about last was ... they had a road check and they were intercepting all the mail that came into the area ... and all the business men and people who had money in the area their mail was taken from them and they were going to read it all out ... the whole thing was that based on this ... (laughs) ... the central characters ... (mumbles) ... you ever heard of the Broons in the Sunday Post? ... anyway it's in there ... and all the characters were around that ... and the fact that ... as they didn't eat these potatoes ... then (?than) some other food (?mumbled) ... they would go to heaven when they died ... and this is ... this is what it was about basically ... it started off first of all I can see it just as a comic strip done in a paper you know ... the father he was eating this ... he was eating a meal then he hurt his back, he was lying down he couldn't move and ... no, sorry ... just remembered now ... the mother she was wanting some help in the house and the father he said he'd hurt his back ... then ... then they had a meal ... anyway it all came round that he hurt his back and ... actually hurt his back when there was nothing wrong with him in the first place, then he did hurt his back and he was lying there ill and the wife (?) she was trying to comfort him ... which is all (?) very sick but it was all this if they didn't eat these mince and potatoes they wouldn't go to heaven ... first of all they had all the poor crawling out walking out to fields to steal potatoes and the people started protecting the potatoes then they had the people crawl along the road to get out to these fields they were crawling along the ground and things and people were feeling sorry for them so they were throwing bits of food to them and throwing them an odd potato ... that's about all there was actually ..."

"Was there anything earlier, before this?"

"No, as I say it just started off reading this cartoon strip in this book ... 'cause they put out an annual every year with cartoon strips in and I was just reading one of these ..."

"Any more details?"

"I'll give you all the characters: there's a mother, a father, two twins ... two twin boys, a young girl, a grandfather, two daughters, two sons ... and ... another studious son, that's it, that's it, eleven of them ... and there was also ... there were

some other people there I didn't recognise ... it was like a play or something, people kept floating out and in doing little bits but I didn't recognise any of the rest of them at all ..."

"Did you see things?"

"Yes, lots and lots of things: it was all really clear ..."

"Did it seem real at the time?"

"It did seem real actually I must admit."

"Would you say you were observing only, or participating?"

"... Seemed to be observing, yes ... more than taking part ..."

"Would you describe it as everydayish, or bizarre or unusual?"

"Really unusual."

"Did you have any feeling or emotion?"

"Yes, I felt quite concerned about these people not being able to get these potatoes, yes, a little bit ..."

"Anything else?"

"No."

"Nothing else at all?"

"No."

"Thanks. Goodnight."

(b) The effect of nitrazepam on dreams

- (i) An everydayish dream on 20 mg nitrazepam. Note that although the content is everydayish, the dreamer says he is dreaming, not thinking and he describes himself as participating.

445

"Could you tell me what was passing through your mind please?"

"I was parking my bicycle outside a shop I think it was a butcher's shop ..."

"Go on."

"I asked somebody to keep an eye on it ..."

"Go on."

"... It's not that I'm falling asleep, I just can't remember ..."

"Was there anything earlier, anything before this?"

"... No I can't remember anything ..."

"Can you give any more details about what you can remember?"

"... No."

"Were you thinking or dreaming?"

"Dreaming."

"Participating or not?"

"Yes."

"Were there any people?"

"There were, I think there were many people but no special people, no individuals - nobody I recognised ..."

"Was there any setting?"

"It seemed that the butcher's shop was in a row of shops which were set back off the road or rather they had their own small road running in front of them set back off the main road ..."

"Was it familiar?"

"No."

"Did you have any feeling or emotion, either pleasant or unpleasant?"

"No."

"Anything else at all you can recall?"

"... No."

"Thanks, goodnight."

-
- (11) Withdrawal of nitrazepam produces bizarre experiences - which may not even be called dreams, however.

433

"Could you tell me what was passing through your mind please?"

"Rather strange. It was about this sort of chain letter thing

(that I got a few weeks ago) ... but its not like a normal one ... it does work, this thing, if its done properly and all that ... and it was about ... this young girl was wandering about in a sort of garland thing and she had a big paper bag full of money and I could hear someone else telling this man about it, it was a new welfare thing or something, he was saying ... and he eventually went up to the young girl and asked her ... and she just emptied out this paper bag and it was just big sweets that came tumbling out sort of thing. Then you woke me up ... yes, that's about it ..."

"Anything before this?"

"Not that I can remember, no ..."

"Can you give some more details about this?"

"... Not really, its all pretty vague, I wasn't participating, I was just hearing it ..."

"Anymore details?"

"... No, can't thing of anything else?"

"Quite sure?"

"... Yes, I'm sure can't think of anything else."

"Would you say you were thinking or dreaming?"

"I don't know, I may have been thinking about this letter thing myself ... yes, I think I was thinking."

"You said you weren't participating?"

"No."

"Have you mentioned all the people?"

"Yes."

"Was there any setting?"

"No, there wasn't any setting."

"Did you have any emotion or feeling, pleasant or unpleasant?"

"No, I didn't have any."

"Anything else at all you can think of?"

"Yes, I didn't know any of the characters involved, didn't know any of them at all ..."

"Anything else?"

"... Not that I can recall ..."

"Quite sure?"

"Yes."

"Thanks, goodnight."

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